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LEPROSY.

LEAGUE OF NATIONS. HEALTH ORGANISATION. **Report on the Study Tour of the Secretary of the Leprosy Commission in Europe, South America and the Far East. January 1929–June 1930.** C.H.887.—48 pp. 1930. Geneva.

——. ——. **The Principles of the Prophylaxis of Leprosy. First General Report of the Leprosy Commission.** C.H.970. L.o.N.P. III. Health. 1931. III. 2.—12 pp. Geneva. [6d. $0.15.]

The report on the tour of the Secretary of the Leprosy Commission, Dr. BURNET, is a mine of information on the prevalence of leprosy in the most infected countries of the world, and the very varied methods of prophylaxis adopted by them, which should be consulted by all interested in the subject. Much of the information has already been dealt with in this *Bulletin*, but the present report will be most useful for reference. The data regarding the prevalence of the disease are summarized in an annex. In a section on conclusions the slow progress in the prophylaxis of leprosy is emphasized and it is stated that "the regulations are still too administrative in tone and have not yet acquired the necessary medical character." The great divergence of views which still persists regarding the value of segregation is pointed out, and a plea is made for meetings between leprologists to discuss a general policy. With regard to treatment it is stated: "this question dominates all others to-day" and early treatment is generally agreed to offer the best chances of success, but grave differences of opinion still remain regarding the duration of the good effects obtained by modern methods. Research and organization are, therefore, still required and prophylaxis should be placed on an international basis.

The first general report of the Leprosy Commission deals with principles of prophylaxis in the light of the great advances made during the last decade as the result of improved treatment, which is now regarded as "the most important line of attack in addition to that of isolation." A diet rich in vitamins, exercise and the use of the ethyl esters and salts of chaulmoogra oils are the special methods of treatment advised. To prevent isolation promoting concealment of cases, with delay in the all-important early treatment, segregation should only apply to infectious cases, with dermatological clinics for early uninfective ones at hospitals, or at special leprosy clinics when

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the cases are numerous. Medical students and practitioners should have special courses of instruction in the treatment at one or more central leprosy institutions in each country, where research will also be carried on. Propaganda is necessary to combat indifference to the disease on one hand, and blind panic on the other, and to teach the people that leprosy is contagious, but also remediable as with tuberculosis. Crippled uninfective patients may require special colonies, and all those apparently recovered should be periodically examined after discharge. The children of lepers should be separated from the parents as soon as possible after birth, and marriages of lepers and propagation of children by them should be discouraged. School children should be inspected and all leprous ones removed. The conclusion is also come to that "there is no reliable system of prophylaxis without treatment, and it is generally accepted that the earlier the treatment is instituted the better will be the results." It is much to be desired that these propositions should be accepted and acted on in all leprosy-infected countries.

L. Rogers.

LEPROSY REVIEW. 1931. July. Vol. 2. No. 3. pp. 79–118. With 8 figs. on 4 plates. Quarterly Publication of the British Empire Leprosy Relief Association, 29, Dorset Square, London, N.W.1.

This number contains a review of the Leonard Wood Memorial Conference on Leprosy by T. F. G. MAYER, articles on the intradermal injection treatment by the Editor and on the leprosy problem in Cebu, Philippines, by J. RODRIGUEZ, on non-irritating iodized ethyl esters of *Hydnocarpus wightiana* by H. I. COLE, chemist in the colony at Culion, and an abridged memorandum on the present position of prophylaxis against leprosy in relation to recent improvement in treatment. In Cebu 5,290 lepers were discovered between 1902 and 1929, and most of them were sent to Culion. A close relationship was demonstrated mathematically between density of population and leprosy prevalence. A leprosy dispensary has recently been opened in Cebu for the treatment of early cases.

L. R.

LEPROSY REVIEW. 1931. Oct. Vol. 2. No. 4. pp. 120–162. With 7 figs. (1 map) on 2 plates. Quarterly Publication of the British Empire Leprosy Relief Association, 29, Dorset Square, London, N.W.1.

This number opens with an article by E. BURNET on the League of Nations and the fight against leprosy on the lines of the first article reviewed in this number of the *Bulletin*. A. C. Stanley SMITH deals with leprosy in Kigezi, Uganda, where an island settlement is about to be started: the disease is of a severe type. R. G. COCHRANE reports what is known of the prevalence of the disease in Kenya, Zanzibar and Tanganyika, and he pleads for a whole-time leprosy worker to carry out a survey in the last-named area. F. G. ROSE reports favourably on the treatment of leprosy in the Mahaica leprosy hospital of British Guiana, where the proportion of patients discharged without deformity has risen from 14·3 per cent. in 1923 to 93·8 per cent. in 1930. Dr. Isabel KERR of Dichpali deals with metabolism in relation to treatment, and advises gr. ⅛ of P.D. & Co. thyroid extract daily in cases with alopecia of the eyebrows, ichthyosis and scaly lesions. A. R. DAVISON

of the S. Africa Emjanyaya Leprosy institution writes: "Scoffers at the efficiency of our modern anti-leprotic treatment are as plentiful in South Africa as they are in other parts of the world," and records that prior to regular treatment being begun in 1926 only burnt-out crippled uninfective cases were discharged, but from 1927 to 1930 active treatment increased the discharge rate two and a half fold, mostly uncrippled patients. He advocates 5 cc. doses of ethyl esters or 5 per cent. alepol, especially the latter, 100 cc. of a 10 per cent. solution being non-toxic but painful.

L. R.

British Empire Leprosy Relief Association. (Indian Council.) **Report 1930** [Smith (H. Moncrieff), Chairman.]—112 pp. With 12 figs. (3 maps, 1 folding) & 1 folding chart.

This report gives a good account of the propaganda, survey and treatment plan of work in India. Surveys reveal from two to five times as many cases as are returned in the census figures, and also lead to the establishment of leprosy clinics in which very numerous early amenable cases are treated at small cost. Research work was continued at the Calcutta School of Tropical Medicine, where 104 doctors were also instructed in the treatment of the disease, and 23 more at the Dichpali Leprosy Hospital in Western India. A good example of nine infections through the Hindu joint family system of several generations of a family living together is recorded. A survey of the Almora district of the Kamaon Himalayas showed the main distribution of the disease in villages along the main roads, and infection has been traced to the use by others of blankets of lepers. Clinics are being started at all the district dispensaries. In Bihar and Orissa 9 new clinics were added in the year to the 19 already existing and 5,465 leper patients were treated regularly in the asylums and special clinics in addition to those attending the general hospitals and dispensaries. In the Central Provinces 32 new leprosy clinics were established and no less than 26,564 lepers were treated during 1930, or three times the total in all the leper institutions of the whole of India a few years ago. In Madras the cost of treatment per patient per annum came to 4 shillings.

L. R.

de Mello (I. Froilano). Comment nous avons cherché à résoudre le problème de la lèpre dans l'Inde Portugaise. [**The Leprosy Problem in Portuguese India.**]—*Rev. d' Hyg. et de Méd. Préventive.* 1931. May. Vol. 53. No. 5. pp. 321–341.

The author has interested himself in this question since 1916 when the cases were estimated at 100 in Goa, but a recent search has revealed 220 lepers, half of whom are mixed cases and 46 early ones. For the latter only temporary isolation with effective treatment until fit for discharge is advocated with subsequent watching, while cases with only early nerve symptoms may be treated at hospitals and dispensaries. Healthy contacts should be watched for five years and treated directly the earliest signs of the disease appear. A central leprosy institution will be established in the country with humane and scientific rules on the lines of the laws of Norway and Brazil. A draft of the necessary legislation is appended to the paper.

L. R.

TISSEUIL (J.). De la diffusion du bacille lépreux dans un milieu à lèpre endémique. [**Diffusion of Leprosy in an Endemic Area.**]—*Bull. Soc. Path. Exot.* 1931. June 10. Vol. 24. No. 6. pp. 458–463. [Gaston Bourret Inst., Nouméa.]

It has been suggested by MARCHOUX that leprosy may in some places be as widespread as tuberculosis, and this question has been investigated during a visit to 20,000 natives in New Caledonia. Cases were classified into those positive bacteriologically, those with definite symptoms of leprosy, but negative on examination, and those with doubtful symptoms. Among 1,409 people examined out of a population of 1,852 there were found 25 positive, 90 suspected and 313 doubtful, or a total of 22·2 per cent., but in persons examined at a laboratory the total doubtful was 35·7 per cent. The conclusion is come to that in the absence of a reliable biological reaction the number of possible lepers may be fixed at about 30 per cent., so the isolation of 1 to 3 per cent. is not an effective form of prophylaxis, since those remaining free keep up the disease. Hygienic education is advocated as the basis of all prophylaxis.

L. R.

LAQUIÈZE. Prophylaxie de la lèpre en Nouvelle-Calédonie et dépendances. [**Prophylaxis of Leprosy in New Caledonia.**]—*Bull. Soc. Path. Exot.* 1931. June 10. Vol. 24. No. 6. pp. 483–496.

This paper gives further detailed figures of the prevalence of leprosy in this French colony ; there is a fall of definite cases from 877 in 1915 to 666 in 1930, but a rise of suspected cases from 378 to 1,367. In the sanatorium of Ducos there are 129 cases. Only 27 cures were reported since 1913 with 8 relapses. No treatment is practicable in the village isolation institutions, where the conditions are not satisfactory.

L. R.

HASSELMANN-KAHLERT (Margarete). Das Problem der Lepra auf den Philippinen. [**The Problem of Leprosy in the Philippines.**]—*Deut. Med. Woch.* 1931. Apr. 24. Vol. 57. No. 17. pp. 724–727.

This is a general account of the well-known work in the Philippines. The figures quoted go as far back as 1927. The most interesting point brought out is that the children of lepers are now separated from their parents after six months, and if they remain free from all symptoms for two years they are sent to their parents or to an education establishment near Manilla. In 1928 10–15 per cent. of the children of the lepers became infected through contact with their mothers before separation at the age of six months.

L. R.

AGUESSY (D.). Sur la fréquence de la lèpre au Haut-Dahomey. [**Leprosy in Upper Dahomey.**]—*Bull. Soc. Path. Exot.* 1931. July 8. Vol. 24. No. 7. pp. 585–587.

In the course of work on trypanosomiasis the author examined 8,944 of the 41,233 inhabitants of the Djougou circle of Upper Dahomey

for leprosy and recognized 130 cases, or 14·5 per mille, which he considers to be far below the true number. In other regions the people fear the disease and isolate cases strictly when they are recognized, but in Dahomey infected persons remain free in the families and are even readily married; this accounts for the high incidence.

L. R.

SPOTO (Francesco) & RUFFINO (Paolo). Contributo allo studio della lebbra familiare. [**Familial Leprosy.**]—*Arch. Ital. Sci. Med. Colon.* 1931. May 1. Vol. 12. No. 5. pp. 271–280. With 6 figs. (1 coloured on plate). English summary (3 lines). [Inst. of Colonial Path., Univ., Modena.]

The authors record 4 cases of leprosy in the same family, a mother and 3 children, the latter of whom had always lived in Italy. The mother, 32 years of age, came from S. Paulo, Brazil, at the age of 4 years, and did not develop signs of leprosy till after 18 years' residence in Italy. The children affected were a girl of 16 years, and 2 boys of 11 and 10. The mother and daughter presented the mixed form and bacilli were present in the nasal mucus; the two sons were in an early stage of the nodular form, and so far the bacilli had not been found. It is presumed that the mother contracted her infection in S. Paulo and the children from her by direct contact.

H. H. S.

USHER (B.). **A Case of Leprosy.**—*Canadian Med. Assoc. Jl.* 1931. May. Vol. 24. No. 5. pp. 693–694. With 2 text figs. [1 ref.] [General Hosp., Montreal.]

The author reports a case of nodular leprosy seen in Canada. The patient is considered to have become infected while in charge of Chinese in France during the great war. A service pension has been granted to him.

L. R.

TISSEUIL (J.). Contribution à l'étude d'un test thermique de guérison clinique de la lèpre. [**The Body Temperature as a Test of Recovery in Leprosy.**]—*Bull. Soc. Path. Exot.* 1931. Apr. 15. Vol. 24. No. 4. pp. 272–274. [Gaston Bourret Inst., Nouméa.]

Three cases of leprosy are recorded in which the symptoms had apparently disappeared, but irregularities in the temperature not explained by any other cause, such as tubercle, raised a suspicion that the leprosy had not been cured, so it is suggested that this sign may indicate that cutaneous symptoms may not remain long absent.

L. R.

LE GAC (P.). Fréquence du signe de la percussion douloureuse dans la lèpre. [**The Painful Percussion Sign in Leprosy.**]—*Bull. Soc. Path. Exot.* 1931. May 13. Vol. 24. No. 5. pp. 363–366.

The symptom of pain on percussion was first described in leprosy by LE DANTEC in New Caledonia in 1889. It is elicited by percussing with the fingers when acute pain may be produced in areas affected by leprosy in the absence of visible skin lesions, especially over bones,

and later over tendons, ligaments and other tissues. It may occur as hyperaesthesia in areas which subsequently become anaesthetic. The author observed this sign in 70 per cent. of 133 cases examined, most frequently in nodular and mixed cases and least so in macular cases.

L. R.

TOLOSA (Adherbal). Les névrites lépreuses. [**Leprous Neuritis.**]—*Rev. Sud-Américaine de Méd. et de Chirurg.* Paris. 1931. June. Vol. 2. No. 6. pp. 593–603. With 4 text figs. [14 refs.]

The difficulties in the diagnosis of some forms of nerve leprosy are discussed in this paper on general lines as well as the pathology of nerve leprosy. Serial sections indicate that the propagation of the disease is from the periphery upwards in the nerves, but isolated lesions in the nerves are of circulatory origin. A case of leprous paralysis of the inferior laryngeal nerve is mentioned. When simple laboratory examinations give negative results sections of the tissues may clinch the diagnosis.

L. R.

VILDE (J.). Histopathologische Untersuchungen ueber das Zentralnervensystem der Leprakranken. [**Histopathology of the Central Nervous System in Leprosy.**]—*Ztschr. f. d. gesamte Neurol. u. Psychiat.* 1931. Apr. 18. Vol. 133. No. 1/2. pp. 119–136. With 3 text figs. [Refs. in text.] [German Research Inst. for Psychiatry [K. Wilhelm Inst.] Munich.]

Thinking that too little attention has been paid to the secondary effects of leprosy on the central nervous system the author has examined the brain in fifteen cases, two of which with basal meningitis and one with sepsis were excluded. In the remainder he found no primary lesions due to leprosy, but he noted secondary fatty and arteriosclerotic degenerations of the arteries in some cases. The inner membranes of the brain also showed fibrous thickening, and degenerative and occasionally inflammatory lesions of the parenchyma were observed, but these also were not peculiar to leprosy. They showed themselves chiefly in vacuolation of the ganglion and glia cells. No leprosy bacilli were found in the brains, so the lesions described are attributable to toxic action.

L. R.

MUIR (Ernest). **The Treatment of Leprosy.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1931. Aug. 8. Vol. 25. No. 2. pp. 87–94. [School of Trop. Med. & Hyg., Calcutta.]

Muir's well-known methods of treatment are described. The main point on which he now lays stress, in addition to those formerly advocated by him, is the value of intradermal infiltration of the skin lesions with hydnocarpus esters. He uses for this purpose the ethyl esters washed with steam and iodized to remove their irritating properties as made in the Philippines. From 10 to 20 square inches of skin may be infiltrated by as many as eighty to one hundred punctures with a short guarded needle at one sitting, with the injection of 5 cc. of the ester.

L. R.

PANETH (O.). Zur Chemotherapie der Lepra. [**Treatment of Leprosy.**] —*Arch. f. Schiffs- u. Trop.-Hyg.* 1931. Aug. Vol. 35. No. 8. pp. 467–470. [7 refs.]

The author records his experience in Sumatra with the use of thymol, krysolgan, sanocrysin and chaulmoogra derivatives. The first three he found to be useless, but chaulmoogra ethyl esters in the form of antileprol was more effective in early cases treated for long with large doses. Unfortunately the early cases tend to be hidden until too late to get the best results. On theoretical grounds he suggests the use of silver preparations ; these he is now testing.

L. R.

DIKSHIT (B. B.) & ROW (Rao Sahib T. Madhava). **A Preliminary Note on the Actions and Uses of "Alepol."**—*Indian Med. Gaz.* 1931. June. Vol. 66. No. 6. pp. 317–320. With 6 figs. on 1 plate. [2 refs.] [Med. College, Vizagapatam.]

The authors found alepol unirritating to the tissues, while rabbits could tolerate at least 25 mgm. per kilo and cats showed no appreciable effects after 150 mgm. per kilo hypodermically. Orally it had some irritant action on the gastro-intestinal tract, and intravenously it lowered the blood pressure and it has marked haemolytic properties. In leprosy it gave very good results in some cases, but no improvement occurred in others; a statement based on only 9 cases treated during seven months.

L. R.

NOLASCO (J. O.). **Local Effects of Infiltration with Iodized Ethyl Esters of Hydnocarpus wightiana Oil in Non-Lepers.**—*Jl. Philippine Islands Med. Assoc.* 1931. June. Vol. 11. No. 6. pp. 219–225. With 3 text figs. [1 ref.]

The effects of infiltration of the skin of non-lepers with hydnocarpus esters have been studied by histological examination of the treated tissues excised after twenty and thirty-four days respectively. Definite cellular reaction with large mononuclear cells loaded with yellowish globules were found, which can persist over nine months, but were absent in control injections with salt solutions. The sebaceous glands became atrophied in areas infiltrated for seven and a half months.

L. R.

STEIN (A. A.). Ueber die Lokalbehandlung der Lepragesschwüre. [**Local Treatment of Leprous Ulcers.**]—*Dermat. Woch.* 1931. Aug. 22. Vol. 93. No. 34. pp. 1345–1347. With 2 text figs. [2 refs.]

Working in Russia the author reports good effects in healing old leprous ulcers on the legs by applying poultices with 10 per cent. magnesium sulphate to reduce the pus formation, followed by an

ointment containing 10–25 per cent. of the same salt. Healthy granulations soon appear followed by healing, which lessens the danger of secondary septic infections.

L. R.

MENAUT. Le traitement de la lèpre d'après la méthode de Pen. [**Treatment of Leprosy by Pen's Method.**]—*Bull. Soc. Méd.-Chirurg. Indochine.* 1930. Aug.–Sept. Vol. 8. No. 8/9. pp. 799–805.

In 1907 a leprosy centre was discovered in a forest area in Cambodia, where a special treatment was carried out by Kruv PEN, who was illiterate. The author found the medicine contained sixteen ingredients, including Krap-Krabao, the local name for *Hydnocarpus anthelmintica*, which was doubtless the active principle. Certain substances, including copper sulphate, were also applied externally. The degree of benefit resulting is uncertain.

L. R.

GOHAR (M. A.). **Vaccine Treatment of Leprosy.**—*Jl. Trop. Med. & Hyg.* 1931. June 15. Vol. 34. No. 12. pp. 166–168. [11 refs.] [Bact. Dept., Faculty of Med., Cairo.]

The author reports favourable effects in nodular leprosy only following the injection of bacillary suspensions made from nodules, together with other saprophytic organisms cultivated from nodules.

L. R.

RUBINO (M. C.). Séro-diagnostic de la lèpre par l'agglutino-sédimentation des globules de mouton formolés. [**Serodiagnosis of Leprosy.**]—*Ann. Inst. Pasteur.* 1931. Aug. Vol. 47. No. 2. pp. 147–172. With 1 text fig. [16 refs.]

——. Séro-diagnostic de la lèpre par la agglutino-sédimentation des globules rouges de mouton formolés.—*Bull. Acad. Méd.* 1931. June 2. Year 95. 3rd Ser. Vol. 105. No. 21. pp. 890–893.

This is a comprehensive paper on Rubino's agglutination-sedimentation test with formolized sheep's red corpuscles in which minute details of the technique are given and the results of an investigation in MARCHOUX's laboratory in Paris are recorded. He comes to the following conclusions. The sensitiveness of the reaction was clear and it was never obtained in any condition except leprosy, for the serum of lepers contains a specific substance which causes rapid agglutination, and sedimentation of formolized sheep's red corpuscles. Certain human sera contain hetero-agglutinins sedimenting indifferently natural and formolized sheep's corpuscles. These can be removed by the use of unformolized corpuscles. A reaction is positive if sedimentation is produced in less than an hour with the formolized corpuscles or occurs to a much greater degree with them than in control tubes, but is otherwise negative. In 36 cases of leprosy 27 were positive, 7 negative and 2 doubtful, but all of 304 sera from various other diseases were negative, with one exception in a patient with thickened nerves who ultimately was found to have nerve leprosy. The second paper is a brief account of the same work.

L. R.

ADANT (Max). La réaction de Botelho dans la lèpre. Technique d'Itchikawa et Baum. [**The Reactions of Botelho and Rubino in Leprosy.**]—*C.R. Soc. Biol.* 1931. June 30. Vol. 107. No. 21. pp. 907–908. [4 refs.]

——. La réaction de Rubino dans la lèpre.—*Ibid.* pp. 909–911. [10 refs.] [Bact. Lab., Elisabethville, Belgian Congo.]

RUBINO (C.). Séro-diagnostic de la lèpre par l'agglutino-sédimentation des globules rouges de mouton formolés. A propos d'une note de M. Max Adant.—*Bull. Soc. Path. Exot.* 1931. July 8. Vol. 24. No. 7. pp. 519–521. [1 ref.]

In the first of these notes the author obtained 10 positive reactions in 14 lepers, or 71·4 per cent., and he quotes LE GAC's 72 per cent. and da Silva ARAUJO's 50 per cent. of positive results of their tests. Among 138 non-lepers all were negative. In the second note he records his results with Rubino's test with formolized corpuscles and he reports 9 positive reactions in 14 negro lepers, or 64 per cent.

In a note on the papers of Adant Rubino points out that the results of the former are not comparable with those of Rubino's new technique.

L. R.

LANDEIRO (Fausto). Sédimentation globulaire dans la lèpre. [**Red Cell Sedimentation in Leprosy.**]—*Arquivos Inst. Bact. Camara Pestana.* 1930. Vol. 6. No. 2. pp. 167–171. With 1 chart in text. [7 refs.] [Camara Pestana Inst., Lisbon.]

This paper deals with the simple red corpuscle sedimentation rate in citrated blood. The author examined the blood of 50 lepers and he obtained sedimentation rates of 68 in nodular, 26 in nerve and 56 in mixed cases of leprosy. He agrees with other workers that the rate is much higher in leprosy than in healthy persons, and is especially high in the nodular type.

L. R.

GOMES (J. M.). Denuncia serologica da lepra latente. [**Unmasking of Latent Leprosy.**]—*Brasil-Medico.* 1931. July 18. Vol. 45. No. 29. pp. 661–663. [Hyg. Inst., São Paulo.]

The author has for several years performed the test of complement fixation in patients suspected of leprosy infection, using de-fatted Deycke's bacillus as antigen. He speaks very highly of its reliability. Some cases of active tuberculosis give a positive reaction, and a few "regarded as leprosy on account of a positive reaction proved to be latent tuberculosis." If, however, the preparation of the antigen be very carefully carried out, only the former (active tuberculosis) will mar the value of the test and then very occasionally. Of 148 contacts 63 reacted positively (42·5 per cent.) and after taking potassium iodide, 2 gm. daily for a week, another 16 previously negative became positive (53·3 per cent. in all). Brief notes are given of 13 patients, all of whom had shown signs of the disease in the past, from 3 months to 4 years 8 months previously. None of these showed bacilli on examination of the nasal mucus; 9 were positive on testing the serum direct for complement fixation, and the other 4 were also positive after taking the potassium iodide (one giving ++++). One patient, a man of 24 years, had showed slight signs 52 months before (his sister was leprous) and his serum gave ++, reduced to + after KI; this anomalous

result is to be the subject of a special communication. The author records these cases as a plea for systematic examination by complement fixation of contacts and suspects presenting no obvious signs, and, if the results are negative, a retest after administration of potassium iodide before a final opinion is expressed.

H. H. S.

ALI (Mohamed) & EL-DALGAMONY (A.). **The Result of the Seriological** [sic] **Examination of 257 Lepers and 527 Non-Lepers for Syphilis by the Wassermann's and the Kahn's Flocculation Tests.**—*Jl. Egyptian Med. Assoc.* 1931. June. Vol. 14. No. 6. pp. 394–399. [22 refs.]

After references to the literature, and pointing out that the Kahn flocculation test seems to give a negative reaction in the sera of non-syphilitic lepers which are otherwise positive by the Wassermann test, they record the results by both tests with the sera of 257 lepers and 527 non-lepers. They found that the Kahn test gave less positive reactions in leprosy than the Wassermann and in non-leprosy cases the opposite result was observed.

L. R.

BADGER (L. F.). **Significance of Positive Wassermann and Kahn Reactions in Leprosy.**—*Public Health Rep.* 1931. Apr. 24. Vol. 46. No. 17. pp. 957–970. [24 refs.]

After references to earlier literature tests with the sera of 207 lepers over ten years of age with Kolmer's antigen for the Wassermann and Kahn's for his test are dealt with. The author concludes that positive reactions were obtained in an abnormally high proportion of the cases, nearly twice as frequently in females as in males and more often in patients under than over twenty. Plus reactions were three times as high in lepers as in a control group and definite changes in the serum reaction were correlated with clinical variations in the leper patients.

L. R.

HÉRIVAUX (A.). Les groupes sanguins dans la lèpre. [**Blood Groups in Leprosy.**]—*Bull. Soc. Path. Exot.* 1931. July 8. Vol. 24. No. 7. pp. 618–619. [Pasteur Inst., Antananarivo.]

The blood of 78 patients with nodular or nerve leprosy were examined in Madagascar and fewer lepers than normal were found to belong to group AB, but more to groups B and O.

L. R.

i. OTA (M.) & SATO (S.). Culture de deux variétés d'un bacille acido-résistant à partir du sang et du léprome de lépreux (*Mycobacterium aurantiacum* et *M. album*). [**Cultures from Lepers.**]—*C.R. Soc. Biol.* 1931. July 16. Vol. 107. No. 23. pp. 1062–1063.

ii. —— & ——. Culture du bacille tuberculeux à partir d'un léprome typique et d'un ganglion lymphatique de lépreux.—*Ibid.* pp. 1064–1065. [1 ref.] [Dermat. Clinic, Tohoku Imperial Univ., Sendai, Japan.]

i. In the first of these short notes the authors report having made cultures from the blood of 54 lepers on media suitable for the growth

of acid-fast organisms. Half of the patients were also suffering from tuberculosis. No tubercle bacilli were obtained, but from two acid-fast bacilli named *Mycobacterium aurantiacum* and *M. album* were grown. On inoculating white rats with these organisms swelling and inflammation of the nose was produced with discharge of masses of acid-fast bacilli, so they consider them to be lepra bacilli.

ii. In the second note they record the cultivation of Koch's tubercle bacillus from a typical leprous nodule, which on injection subcutaneously into guineapig produced necrosis of the tissues. They also obtained the tubercle bacillus from the axillary gland of a leper who was suffering from pulmonary tuberculosis.

L. R.

HENDERSON (John M.). **Preliminary Observations on an Acid-Fast Organism isolated from Human Leprous Lesions.**—*Indian Jl. Med. Res.* 1931. July. Vol 19. No. 1. pp. 145–153. With 7 coloured figs. on 2 plates. [3 refs.] [School of Trop. Med. & Hyg., Calcutta.]

This is an account of an acid-fast bacillus isolated by E. MUIR from a case of leprosy by Shiga's method of culture, which only showed visible growth eight months after insemination on the culture tubes. It now grows readily on Petroff's medium and on glycerine broth under partially anaerobic conditions as a yellow film, but growth is most abundant in the presence of oxygen. Stained by Ziehl Nielsen's method it shows beaded red rods, but if first treated with zylol and absolute alcohol many of the rods stain blue. Attempts to demonstrate the presence of bacterial antibodies in the sera of lepers failed, but this does not prove that it is not the organism of leprosy. It was only obtained by MUIR once in twenty-three trials from human leprous tissues, and the conclusion is come to that "its relationship to M. leprae (Hansen) is at the moment completely undetermined."

L. R.

NINNI (C.) & MONALDI (T. de Sanctis). Formes cachées des tuberculosis dans la lèpre humaine. [**Larval Forms of Tuberculosis in Leprosy.**]—*C.R. Soc. Biol.* 1931. July 3. Vol. 107. No. 22. pp. 981–983. [1 ref.]

In view of the frequency with which lepra bacilli occur early in leprosy in the lymphatic glands the authors have tried injecting leprous material directly into the glands of guineapigs. On examining the animals killed after various periods of time they found only enlargement and congestion after 25 days, some points of necrosis after 45 days and enlargement and caseation at the end of 60 days, but only slight enlargement after 90 days. Acid-fast bacilli were numerous after 4 days, but gradually decreased and disappeared in 90 days. From the 25th to the 60th day the organs of the inoculated animals showed typical tubercular lesions, but no such lesions were found after 90 days, and human tubercle bacilli were cultivated in the earlier periods only.

They conclude that the inoculation of apparently pure leprous material into the glands of guineapigs produced an atypical tuberculosis in the animals, such as is frequent in leprosy.

L. R.

BASOMBRÍO (Guillermo A.). Estudio de las adenopatías en la lepra.—*Semana Méd.* 1931. May 7. Vol. 38. No. 19 (1947). pp. 1213–1234. With 8 text figs. [42 refs.]

KIRK (N. T.). Neurotrophic Changes in Leprosy.—*Milit. Surgeon.* 1931. July. Vol. 69. No. 1. pp. 18–25. With 6 text figs. [2 refs.]

TISSEUIL (J.). Au sujet d'un cas de léprides à forme d'érythème noueux.—*Bull. Soc. Path. Exot.* 1931. May 13. Vol. 24. No. 5. pp. 366–369. [1 ref.]

TISSEUIL (J.). Stade tuberculoïde intermédiaire dans l'évolution d'un cas de lèpre.—*Bull. Soc. Path. Exot.* 1931. June 10. Vol. 24. No. 6. pp. 453–458. [Gaston Bourret Inst., Nouméa.]

TOULLEC & RIOU. Un cas de lèpre atypique avec prédominance des lésions cutanées.—*Marseille-Méd.* 1931. Mar. 25. Vol. 68. No. 9. pp. 414–416.

VAN DEN BRANDEN (F.). Premier cas de lèpre diagnostique au Congo Belge chez un européen.—*Ann. Soc. Belge de Méd. Trop.* 1931. Mar. 31. Vol. 11. No. 1. pp. 9–10.

TROPICAL OPHTHALMOLOGY:

A REVIEW OF RECENT ARTICLES.—XVI.*

CONJUNCTIVA.—RAGAIN[1] found that in Cambodia acute infectious inflammations of the conjunctiva occur with the greatest frequency at the commencement of the rainy season and are specially common throughout its duration. The peak incidence is seen in June. For this distribution he blames flies, pollen and wind. Children are the chief sufferers and Koch-Weeks infection is the most common cause (56 per cent.). Even simple and benign cases may be complicated by the injudicious application of "native remedies." In an interesting paper TALBOT[2] has described seasonal epidemics of gonorrhoeal ophthalmia which occur in the oasis of Gabès, south of Tunis. These epidemics appear in autumn at the time when nomadic merchants and labourers reinforce the local population in order to gather the olive and date crops. Such nomads constitute the source of infection. Flies apparently play no part in the dissemination of the disease which seems to be conveyed directly from person to person. The author refers to his experience of similar epidemics. One of these occurred in Tonking in 1917 amongst Annamite and Tonkinese recruits who were collected in camp previous to embarkation for the Western front. Many cases of partial and of total blindness resulted, but the unfortunate sufferers were considered to have inoculated themselves purposely to avoid service and were discharged without pension or compensation.

Trachoma.—OLITSKY and TYLER[3] report two instances of the experimental direct transmission of trachoma to normal monkeys (*Macacus rhesus*) by gently rubbing the conjunctiva with swabs containing fresh secretion taken from a trachomatous patient. The infected monkeys presented typical granulomatous changes after the lapse of thirteen days. Tissues taken from one of these monkeys yielded cultures of *B. granulosis*. The same bacterium was isolated from the conjunctival tissues of the patient from whom this monkey was infected, and cultures of it injected into three other monkeys (*Macacus rhesus*) induced the changes of experimental trachoma in from nine to eleven days.

MACCALLAN[4] has contributed a valuable paper on the disease. He states that the most reliable method of arriving at the incidence of trachoma in a more or less trachomatized country is a systematic inspection of the primary schools. In 1914 he found in Egypt 92 per cent. of scholars infected. Such conditions, however, may not in every case represent the true incidence of the disease in the country or town in which the examination was made, since the majority of cases tend to cicatrize and after some years it may prove difficult to detect evidence

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* For the fifteenth of this series see Vol. 28, pp. 475–481.

1 RAGAIN (L.). Les conjonctivites infectieuses au Cambodge.—*Bull. Soc. Méd.-Chirurg. Indochine.* 1931. Apr. Vol. 9. No. 4. pp. 247–249. With 1 graph & 1 chart. [Ophthalmic Inst., Phnom-Penh.]

2 TALBOT. Conjonctivites saisonnières épidémiques à gonocoques dans l'oasis de Gabès (Sud tunisien).—*Arch. Inst. Pasteur de Tunis.* 1930. Dec. Vol. 19. No. 4. pp. 451–460. [1 ref.]

3 OLITSKY (Peter K.) & TYLER (Joseph R.). Direct Transmission of Human Trachoma to the Monkey.—*Science.* 1930. May 30. Vol. 71. No. 1848. p. 564. See also *Studies from Rockefeller Inst. Med. Res.* 1931. Vol. 75. pp. 1–2. [3 refs.]

4 MACCALLAN (A. F.). The Epidemiology of Trachoma.—*Brit. Jl. Ophthalm.* 1931. July. Vol. 15. No. 7. pp. 371–411.

of the pre-existing disease. [It should be remembered, too, that the diagnosis between trachoma and folliculosis and other abnormal conjunctival conditions may prove particularly difficult in young subjects.] As a source of infection a trachomatous child is more dangerous than a trachomatous adult. Treatment in schools is the most important agent in prophylaxis as it renders the pupil non-contagious. The author comments on the curiously uneven distribution of the disease; thus, the inhabitants of Mecca are comparatively free, though their neighbours in Nejd are heavily infected. There is practically no rain in Mecca and absolutely no cultivation. In Nejd rain is prevalent and cultivation extensive. In Egypt a rise of conjunctival disease coincides very accurately with a rise in climatic temperature. Most of the ophthalmia is gonococcal, usually non-venereal. A vast increase in the number of flies occurs at the same time. An increase in the patients requiring treatment for trachoma soon follows the epidemic. People living in the neighbourhood of the site of an ancient ruined city, where there is considerable fine dust formed from crumbling bricks, suffer more severely than those who are exposed to the irritation of the coarser desert sand.

WILSON,[5] too, has published an informative paper on certain aspects of the aetiology of the disease. He remarks upon the difficulty of securing a universally accepted definition of trachoma. There exists in all cases of trachoma a stage during which it is impossible to state the diagnosis definitely because the appearances are simply those of any ordinary simple chronic conjunctivitis. As regards the causative organism, interpretations of positive inoculations in animals are particularly open to criticism. In the light of recent research one should not entirely disregard the aetiological relationship of inclusion bodies to the disease; these may indicate the presence of a virus in their immediate vicinity. With respect to *B. granulosis* he and his fellow workers in Egypt have only obtained negative results although they followed closely the technique recommended by NOGUCHI. He thinks that the evidence in favour of this organism being the cause of trachoma is not convincing at present. But its discovery, when we understand the conditions better, may yet prove the key which will open the way to an accurate knowledge of the disease and its remedy.

WEISS[6] succeeded in isolating from trachomatous tissues a small bacillus which corresponded to the description by NOGUCHI of the *B. granulosis*. A subconjunctival inoculation of the cultures in two monkeys (*Macacus inuus*) produced fine granulations after the lapse of fourteen days. Thirty-three days later the symptoms began to disappear. A subculture from one of NOGUCHI's original preparations failed to infect three human volunteers and one monkey. OLITSKY, KNUTTI and TYLER[7] have induced a characteristic granular conjunc-

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[5] WILSON (Rowland P.). A Discussion on the Aetiology of Trachoma with Special Reference to Bacterium Granulosis (Noguchi).—*Brit. Jl. Ophthalm.* 1931. Aug. Vol. 15. No. 8. pp. 433–446. [43 refs.]

[6] WEISS (Charles) with the Collaboration of O. Reymond DE GENTILE & E. CONSEIL. Lésions conjonctivales, consécutives à l'inoculation de *Bacterium granulosis* (Noguchi).—*Arch. Inst. Pasteur de Tunis.* 1930. Dec. Vol. 19. No. 4. pp. 433–434.

[7] OLITSKY (P. K.), KNUTTI (R. E.) & TYLER (J. R.). Transmission and Cultivation Experiments with Human Trachoma and the Experimental Disease in Monkeys.—*Jl. Experim. Med.* 1931. July 1. Vol. 54. No. 1. pp. 31–40. [7 refs.] [Rockefeller Inst. for Med. Research, New York.]

tivitis in *Macacus rhesus* monkeys by either a single subconjunctival injection or by repeated conjunctival swabbings with material obtained from patients suffering from long-standing trachoma. Pathogenic strains of *B. granulosis* were successfully cultivated from six out of the eleven patients from whom this material was taken, and the organisms were recovered from the infected monkeys. They found that material taken from human trachoma and from monkeys suffering from the experimental disease and cultures of *B. granulosis* all induced in the *Macacus rhesus* the same clinical and pathological changes when injected subconjunctivally. These changes closely resembled the follicular stages of human trachoma.

BELOT[8] has investigated the intradermal reaction to trachoma antigen described by TRICOIRE. He examined 400 trachomatous and 100 non-trachomatous patients; 59 per cent. of the former and 52 per cent. of the latter gave a positive reaction. Its value as a diagnostic aid is therefore not great. The author considers that trachoma is a purely local disease and cannot be expected to produce humoral changes, nor is vaccine therapy likely to prove useful in its treatment. NEUMANN[9] advocates the use of iodine in the treatment of the disease, especially when advanced and complicated by pannus and other corneal troubles. The drug is applied to the everted tarsal and fornical conjunctiva with a wisp of wool twisted on to a glass rod. 10 per cent. tincture of iodine is used and is smeared on until the wool holding the iodine is dry. Excess of iodine is then wiped off with dry cotton-wool and the whole area is smeared with a simple ointment. Reaction reaches its maximum the day after the application and usually is completed by the fourth day. Treatment may then be repeated if desired. Copper stick and conjunctival massage are used in conjunction. EL-KATTAN[10] claims to have obtained good results by injecting a vaccine into the fornix. The vaccine is prepared by repeatedly macerating small pieces of trachomatous tissues in saline. The supernatant fluid obtained from each maceration is mixed and then diluted until its turbidity equals that of a 10,000 million per cc. suspension of staphylococci. The emulsion is heated to 60° C. for one hour and one cc. is then added to fifteen cc. of sterile inactivated horse serum. Half a cc. of this serum is injected every five or six days.

The *Revue Internationale du Trachome* for April 1931 contains a note by MACCALLAN[11] on the small pits which are found at the periphery of the upper portion of the cornea in old trachomatous cases. These were first described by HERBERT in 1904 and MACCALLAN considers them to be peculiar to trachoma. VEJDOVSKY[12] reports favourably upon a modified Denig's operation for trachomatous

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[8] BELOT (R.). Etude critique de l'intra-dermoréaction trachomateuse de Tricoire, basée sur 500 cas.—*Arch. Méd. et Pharm. Nav.* 1931. Jan.-Feb.-Mar. Vol. 121. No. 1. pp. 19–22. [1 ref.]

[9] NEUMANN (J.). Iodine in the Treatment of Trachoma.—*Brit. Jl. Ophthalm.* 1931. Sept. Vol. 15. No. 9. pp. 518–524. [17 refs.]

[10] EL-KATTAN (M. Azmy). An Introduction to Trachoma Therapy with Specific Antigen.—*Jl. Egyptian Med. Assoc.* 1931. July. Vol. 14. No. 7. pp. 405–408. With 3 coloured plates.

[11] MACCALLAN. Fossettes marginales de Herbert et les rosettes cornéennes du trachome.—*Rev. Internat. du Trachome.* 1931. Apr. Vol. 8. No. 2. pp. 53–54. [1 ref.]

[12] VEJDOVSKY (V.). Traitement opératoire du pannus trachomateux par l'implantation de muqueuse buccale (procédé de Denig).—*Rev. Internat. du Trachome.* 1931. Apr. Vol. 8. No. 2. pp. 57–62. [20 refs.]

pannus. The operation consists in performing a peritomy above and in grafting into the wound a strip of buccal mucous membrane. The peritomy incision is carried well down on to the sclera and the graft is stitched with fine sutures to the limbus below and to the bulbar conjunctiva above. [Such a graft should diminish the pressure of the trachomatous lid on the cornea since it must lift slightly the diseased tarsal conjunctiva during movements of the lid.] MIKAELIAN[13] records experiments made with respect to the intradermal reactions of trachomatous and non-trachomatous subjects to cultures of Noguchi's bacterium. No specific reaction was obtained. SCHOUSBOÉ[14] contributes a lengthy paper on the treatment of the disease. His main points are that one should try to avoid the occurrence of excessive cicatrization and to deal appropriately with secondary infections. He dislikes copper in stick form, but uses it freely in solution. He prefers silver nitrate for the papillary varieties of the disease and uses it up to 10 grains to the oz. He has found subconjunctival injections of cyanide of mercury (1–5,000) useful in the treatment of trachomatous keratitis. He realizes the importance of treating any associated syphilitic infection. The issue for July 1931 contains several articles, mostly short. HÜBIN and HUBERT[15] discuss the question of an increase of trachoma in Belgium owing to an influx of infected immigrants. These immigrant workmen are often insanitary in their habits and have proved sources of infection in their lodgings. MORAX[16] states that he has been unable to cultivate the *B. granulosis* from material obtained from trachomatous Algerians. He notes the remarkable absence of any bacterial growth in his cultures provided the patient was free from any secondary infection. MORAX and NIDA[17] found that the *B. granulosis* isolated by LUMBROSO at Tunis corresponds generally in its characters with that of NOGUCHI and OLITZA. SÉDAN[18] reports his experience of Tricoire's intradermal reaction. 40 out of 64 trachomatous patients gave a positive reaction, 14 were doubtful and 10 negative. Tuberculous and syphilitic patients reacted positively.

CORNEA.—*Keratomalacia.*—PUSCARIU and NITZULESCU[19] state they have found but little pigmentation of the conjunctiva in xerosis cases seen at Jassy. Recently, however, noticeable pigmentation in a gipsy led them to examine other gipsy patients, and they found that gipsies usually show a brown-blackish tinge of the portion of the bulbar conjunctiva corresponding to the palpebral aperture. They are inclined

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13 MIKAELIAN (R. C.). Le bacille de Noguchi donne-t-il une intradermo-réaction spécifique chez les trachomateux ?—*Rev. Internat. du Trachome.* 1931. Apr. Vol. 8. No. 2. pp. 64–68.

14 SCHOUSBOÉ. Traitement médical du trachome.—*Rev. Internat. du Trachome.* 1931. Apr. Vol. 8. No. 2. pp. 70–106. [36 refs.]

15 HÜBIN (R.) & HUBERT (J.). Le trachome en Belgique et la main-d'oeuvre étrangère.—*Rev. Internat. du Trachome.* 1931. July. Vol. 8. No. 3. pp. 126–132.

16 MORAX (V.). Recherches bactériologiques sur le trachome en Algérie.—*Rev. Internat. du Trachome.* 1931. July. Vol. 8. No. 3. pp. 133–134.

17 MORAX & NIDA. Etude comparative de quelques souches de bactérium granulosis.—*Rev. Internat. du Trachome.* 1931. July. Vol. 8. No. 3. pp. 136–140.

18 SÉDAN (Jean). Recherches sur l'intra-dermo-réaction du trachome.—*Rev. Internat. du Trachome.* 1931. July. Vol. 8. No. 3. pp. 153–158.

19 PUSCARIU (Elena) & NITZULESCU (Julius). On the Pigmentation of the Conjunctiva in Normal Individuals and in Cases of Keratomalacia in Adults.—*Brit. Jl. Ophthalm.* 1931. Jan. Vol. 15. No. 1. pp. 18–22. [16 refs.]

to believe that the pigmentation in xerophthalmia is due to melanin and is a special reaction, the intensity of which varies according to the particular capacity of the tropho-pigmentary tissue of the race; naturally it is least in the white races.

LENS.—*Cataract.*—PÉRETZ[20] describes some of the difficulties experienced by the surgeon when operating for cataract in Egypt. The prevalence of conjunctival diseases and of dacryocystitis renders measures against wound infection specially difficult and important. Cicatricial changes in the eyelids, blepharophimosis and symblepharon, cause difficulty in obtaining sufficient room. Dense leukomata may interfere with the surgeon's view of the knife whilst making the section and may spoil the effect of a technically successful operation. He states that Egypt is the country of glaucoma and that forty per cent. of aged patients there suffer from glaucoma. The co-existence of glaucoma and cataract is very common; frequent examinations should therefore be made of sufferers from incipient cataract, and a preliminary iridectomy should be performed directly a suspicion of glaucoma is aroused. PERETZ considers the hot season unfavourable for operation and prefers not to operate between June and September. KIRBY and NEWELL[21] have conducted a careful investigation into the efficacy of lens protein therapy in cataract, and were unable to satisfy themselves that the patients so treated were in any way benefited.

FLEMING[22] has described the technique advocated by him for the extraction of senile cataract. Preliminary capsulotomy is performed by opening the lens capsule with a Ziegler's knife. A broad "bridge-flap" of conjunctiva is fashioned and the lens is "dipped" by stroking with an iris repositor the sclera beneath the flap. This instrument is employed to elevate the flap whilst the lens is made to present by curette pressure upon the cornea. The repositor is discarded after presentation of the nucleus and delivery is aided by rotating the lens with a needle. Cortical remnants are washed out and replacement effected by irrigation. Fleming considers it safe to omit an iridectomy in the vast majority of cases operated on by this method.

RETINA.—*Detachment of the Retina.*—DOGGART & SHAPLAND[23] have reported on 75 cases of simple detachment of the retina treated at Moorfields Eye Hospital by Gonin's method. Of the 75 patients operated upon, 24 were discharged with the retina in place and in possession of a full visual field; 12 cases showed some improvement; and 39 were either unchanged or worse. This is an encouraging record for such a desperate disease. Before operating it is essential to locate accurately the hole or holes in the membrane; this requires a full mydriasis so that the extreme peripheral region of the fundus may

20 PÉRETZ (H.). A Short Study of the Complications of Cataract Extraction in Egypt with Some Practical Suggestions.—*Brit. Jl. Ophthalm.* 1931. Jan. Vol. 15. No. 1. pp. 28–33. [3 refs.]

21 KIRBY (Daniel B.) & NEWELL (W. Giles). Investigations of Lens Protein Therapy in Cases of Cataract.—*Trans. Amer. Acad. Ophthalm. & Oto-Laryngology.* 1930. pp. 309–322.

22 FLEMING (Norman). Extraction with Conjunctival Bridge.—*Proc. Roy. Soc. Med.* 1931. Apr. Vol. 24. No. 6. pp. 756–757 (Sect. Ophthalm. pp. 26–27).

23 DOGGART (J. H.) & SHAPLAND (C. D.). Simple Detachment of the Retina. With a Report on 75 Cases treated at Moorfields Eye Hospital by Gonin's Method.—*Brit. Jl. Ophthalm.* 1931. May. Vol. 15. No. 5. pp. 257–271. With 8 text figs. [43 refs.]

be explored. It is necessary to ascertain the distance of the hole from the ora serrata and also the meridian of the globe which passes through the middle of the hole. The ora serrata may be assumed to lie 8 mm. from the limbus and the distance of the hole from the ora can be estimated in disc diameters (1·5 mm.). The Moorfields technique closely follows that of GONIN. Two spots at diametrically opposite points of the limbus which are in line with the estimated site of the hole are tattooed with Indian ink. After cocainization, novocaine and adrenalin is injected beneath the conjunctiva at the place selected for puncture and a knotted guiding thread is passed through the episcleral tissue at that limbal pigment mark which is the further remote from the retinal hole. The conjunctiva is incised at a distance of about 12 mm. from the other pigment mark, Tenon's capsule is opened and the sclera is bared over the site of the proposed puncture. A Desmarres retractor, in the centre of which a hole has been punched, is used to retract widely the conjunctival wound. The guiding thread is passed through the hole in the retractor and is arranged so as to traverse both tattoo marks. Its prolongation will thus cross the site of the retinal hole. The previously estimated distance of the hole from the limbus is measured with callipers and the supposed site is marked on the sclera by one of the points which has been dipped in sterilized Indian ink. Conjunctival sutures are inserted and the guiding thread is withdrawn. An electric cautery at white heat now sears a way through the sclera at the point selected, and, as soon as the subretinal fluid has escaped, the cautery is again raised to white heat, plunged through the opening in the sclera and immediately withdrawn. The average depth of penetration is 1 cm. and the duration two seconds. The conjunctival sutures are quickly tied off and a bandage is applied. The patient is nursed with the head so placed that the situation of the hole is the most dependent part of the eye. Both eyes are bandaged and absolute rest under atropine is maintained for eleven days in successful or hopeful cases.

GENERAL DISEASES.—*Onchocerciasis.*—HISSETTE[24] has published a note on the occurrence of onchocerciasis in certain Congo districts. He found that twenty per cent. of those infected had lost their sight; but he does not mention the pathological changes [probably uveitis] which caused the blindness. The eye troubles were greatly ameliorated by the removal of the filarial cysts.

Trypanosomiasis.—HISSETTE[25] describes the optic atrophy, which, even in the absence of any arsenical treatment, may occur in the course of sleeping sickness. Arsenical treatment is a fruitful cause of the complication. Even partial atrophy causes great loss of visual acuity. In simple forms the disc is blanched and has clean-cut edges, whilst the vessels show little or no shrinking. Softening of the disc edges signifies a previous high intracranial pressure. A progressive contraction of the visual field is a more sure indication of an advance of the disease than any afforded by ophthalmoscopic examination. Patients who are the subjects of any form of intoxication are specially liable to contract optic atrophy. Tryparsamide should be administered, and, if there is any swelling of the disc, spinal puncture should be performed.

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[24] HISSETTE. Sur l'existence d'affections oculaires importantes d'origine filarienne dans certains territoires du Congo.—*Ann. Soc. Belge de Méd. Trop.* 1931. Mar. 31. Vol. 11. No. 1. pp. 45–46.

[25] HISSETTE (Jean). Affections oculaires dans la trypanosomiase.—*Ann. Soc. Belge de Méd. Trop.* 1930. Dec. 31. Vol. 10. No. 4. pp. 423–428.

Special attention must be paid to maintaining the nutrition of the patient. Nitrite of amyl or nitrite of soda may prove helpful.

MISCELLANEOUS.—DEPLANCHE[26] has recorded his experiences of eye disease at Abidjan on the Ivory Coast of Africa. Refraction errors account for the majority of consultations by Europeans, and the author agrees with other observers that the amplitude of accommodation in Europeans resident in the tropics suffers diminution and that symptoms of asthenopia appear in them more readily than when living under the more congenial conditions of a temperate climate. Myopia is rare amongst the native population; hypermetropia and hypermetropic astigmatism according to rule is more common. Trachoma is practically non-existent, but gonorrhoeal ophthalmia, both acute and chronic, is rife. The infection nearly always has a genital origin. Leukomata, which result from gonorrhoeal ophthalmia, smallpox, and leprosy, are prevalent. Iritis usually occurs in an insidious form with cyclitis and choroiditis, and often leads to seclusion of the pupil. Cataract is frequently seen, but is very often traumatic or complicated. Few senile cataracts, favourable in every respect for operation, are encountered. Lachrymal affections account for only a small percentage of the admissions. Leprosy and venereal infections constitute the main causes of the ocular troubles in the district. LEGER[27] quotes HOWARD as estimating that in China half a million persons are totally blind, five million are practically blind and fifteen million suffer from defective sight. Lack of proper hygiene leads to trachoma and other conjunctival diseases. Syphilis, too, is responsible for much blindness, as it causes iritis which leads to pupil occlusion or to secondary glaucoma. In some parts of the country xerosis and keratomalacia are as harmful as trachoma. Smallpox also is a common cause of blindness. Destructive inflammations frequently result from the introduction of a virulent infection during the attempts of unskilled persons to deal with mild ocular disorders.

The Annual Report of the Madras Government Ophthalmic Hospital for the year 1930[28] contains, for various reasons, a less full account than usual of the hospital activities; but, nevertheless, much useful information can be gathered from its pages. Both in-patients (3,720) and out-patients (28,470) showed an increase over the previous year. The total number of cataract operations was 1,676; Barraquer's operation being performed on 218 patients. The "bridge-flap" was adopted in the majority of the 1,327 capsulotomy operations. The work of the refraction room is growing and 3,129 patients were treated for errors of refraction. 105 cases of optic atrophy were examined, and 33 cases of choroidal disease. 2,679 patients were treated for "superficial punctate keratitis." It is stated that this disease has altered in character and that the majority of the patients showed marked conjunctival redness and swelling with involvement of the preauricular gland. Fine keratic precipitates, too, were common, but gross iritis was never found. Mild cases were

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26 DEPLANCHE. Deux années de pratique ophthalmologique en Côte d'Ivoire.—*Bull. Soc. Path. Exot.* 1931. May 13. Vol. 24. No. 5. pp. 406–415. [10 refs.]

27 LEGER (Marcel). Les causes de la cécité en Chine.—*Bull. Soc. Path. Exot.* 1931. Apr. 15. Vol. 24. No. 4. pp. 255–265.

28 MADRAS. Annual Report and Statistics of the Government Ophthalmic Hospital, Madras, for the Year 1930 [WRIGHT (R. E.), Supt.].—26 pp 1931. Madras.

relatively infrequent. Experiments were conducted in the treatment of keratomalacia by concentrated vitamin A. This substance, however, was found to be less efficacious than cod-liver oil. Spring catarrh responded much better to small doses of radium than to large ones. It is noted that propaganda on the prevention of blindness is meeting with appreciation.

The Seventeenth Annual Report of the Ophthalmic Section of the Department of Public Health of the Egyptian Government[29] deals with the year 1929. It records an expenditure of £E83,966, and the steady, progressive decrease of blindness amongst the patients attending hospitals shows that good value has been obtained. The percentage has fallen from 15·6 in 1909 to 8·4 in 1929. Purulent ophthalmia causes 75 per cent. of the blindness, and it is stated that 92 per cent. of the population is affected by trachoma. Glaucoma seems to be met with almost as frequently as senile cataract; but more than half the glaucoma is secondary. The report notes the coincident rise of climatic temperature and of hospital attendances of patients suffering from acute ophthalmias. The gonococcus was found in 38 per cent. of the acute ophthalmias.

H. Kirkpatrick.

[29] Egypt: Ministry of the Interior. Department of Public Health. Ophthalmic Section. Seventeenth Annual Report for the Ophthalmic Section, 1929.—42 pp. With 1 folding map. 1931. Cairo.

MALARIA.

JAMES (S. P.), NICOL (W. D.) & SHUTE (P. G.). **On the Prevention of Malaria with Plasmoquine.**—*Lancet.* 1931. Aug. 15. pp. 341–342.

Ten volunteers, all but one students at St. Mary's Hospital, London, were given 0·02 grams of plasmoquine and, on the following day, shortly after taking a second dose, they were bitten by mosquitoes (*A. maculipennis*) heavily infected with the sporozoites of benign tertian malaria. They continued to take plasmoquine, in doses of 0·02 grams, three times a day for 6 days, including the day on which they were bitten, with the result that not one of them became infected. Four control cases, on the contrary, who had no plasmoquine were all attacked by malaria within 14 days. One of these controls was given quinine prophylactically for 8 days but, as usual, when it was stopped it failed to prevent the attack. The authors conclude that "This experiment, and the 15 equally successful trials which preceded it, prove that plasmoquine effectively prevents mosquito-borne malarial infection among a group of healthy individuals who take the prophylactic doses. Quinine lacks this remarkable property . . . From the results of our previous tests, we know that somewhat smaller doses taken over a shorter period are equally effective."

W. Fletcher.

COVELL (G.). **The Present State of our Knowledge regarding the Transmission of Malaria by the Different Species of Anopheline Mosquitoes.**—*Records of the Malaria Survey of India.* 1931. Mar. Vol. 2. No. 1. pp. 1–48. [7 pages of refs.]

Though many species are possible vectors under certain conditions, yet the principal rôle is played in each locality by comparatively few. Infection under laboratory conditions does not prove that a mosquito is an important carrier in nature. The discovery, in nature, of a scanty gut infection in one out of many mosquitoes examined, is of little importance; the discovery of sporozoites, on the other hand, is highly incriminating [but, see SWELLENGREBEL and DE BUCK, below, p. 1018]. The following is a list of the chief malaria-carrying anophelines of the world:

I. *America.*

NORTH AMERICA.

United States	*quadrimaculatus* (south and east).
	maculipennis (Pacific coast).
Mexico	*albimanus* (coast).
	pseudopunctipennis, *quadrimaculatus* (highlands).

CENTRAL AMERICA AND WEST INDIES.

	albimanus.
	tarsimaculatus.

SOUTH AMERICA.

Guiana	*albimanus.*
	albitarsis.
Venezuela	*albimanus.*
	albitarsis.

I. *America—continued.*

SOUTH AMERICA—*continued.*	
Colombia	*albimanus.*
Ecuador	*albimanus.*
Brazil	*albitarsis.*
	tarsimaculatus.
Argentina	*pseudopunctipennis* (north-west).
	albitarsis (north-east).

II. *Europe.*

All countries where malaria occurs	*maculipennis.*
Balkans	*superpictus.*
Italy and Macedonia	*sacharovi* (*elutus*).

III. *Africa.*

NORTH AFRICA.	
Algeria	*maculipennis.*
	algeriensis.
Tripoli	*algeriensis.**
Egypt	*multicolor.**
	*sergentii.**
	*superpictus.**
	*dthali.**
TROPICAL AFRICA.	
Throughout Tropical Africa	*gambiae* (*costalis*).
	funestus.
Belgian Congo	*marshallii* var. *moucheti.*
	nili.

IV. *Asia.*

Asia Minor, Transcaucasia, and Central Asia	*maculipennis.*
	sacharovi (*elutus*).
	superpictus.
Palestine	*sacharovi* (*elutus*).
	sergentii.
	superpictus.
Arabia (south)	*gambiae.*
	*dthali.**
	*culicifacies.**
Mesopotamia	*superpictus* (north).
	stephensi (south).
India and Ceylon	*culicifacies.*
	stephensi.
	listonii.
	minimus.
	maculatus.
	ludlowii.
	philippinensis.†
Siam	*ludlowii.*
	*maculatus.**
Malaya	*maculatus.*
	umbrosus.
	ludlowii.
Cochin China	*maculatus.**
	*leucosphyrus.**
	*kochi.**

* Suspected to be carriers on epidemiological grounds only.

† This species is considered by some observers to be an important carrier in Bengal and Burma.

IV. *Asia—continued.*

China	*hyrcanus* var. *sinensis.**
	*pattoni.**
	*maculatus.**
	*minimus.**
Formosa	*maculatus.**
	*minimus.**
	hyrcanus var. *sinensis.**
Japan	*hyrcanus* var. *sinensis.**

V. *East Indian Archipelago.*

Dutch East Indies	*ludlowii.*
	maculatus.
	aconitus.
	umbrosus.
	hyrcanus (' sinensis ').
	leucosphyrus.
	kochi (occasionally important).
	punctulatus (eastern islands).
Borneo and Labuan	*ludlowii.*
	*umbrosus.**
	*leucosphyrus.**
Philippine Islands	*minimus.*
	ludlowii.

VI. *Australasia, Melanesia and Polynesia.*

New Guinea	*punctulatus.*
New Britain	*punctulatus.*
	punctulatus var.
	moluccensis.
New Hebrides	*punctulatus.**
North Queensland	*punctulatus.**
	*annulipes.**
	*amictus.**
	*bancrofti.**

W. F.

PELTIER. Rapport sur les questions étudiées par la Commission du Paludisme de la Société des Nations réunie à Alger les 20 et 21 mai 1930. (Extraits.) [**Questions studied by the Malaria Commission of the League of Nations assembled at Algiers, May 20 and 21, 1930.**]—*Ann. de Méd. et de Pharm. Colon.* 1931. Jan.–Feb.–Mar. Vol. 29. No. 1. pp. 171–178.

The deltas of many large rivers are almost free from malaria although anopheles are plentiful and the surrounding country is highly malarious. Professors CANTACUZÈNE and ZOTTA have studied this question in the delta of the Danube, where cattle raising is an important industry. They attribute the rarity of malaria to the zoophilism of *A. maculipennis* in this area, where it is the sole vector. In neighbouring districts, in which malaria is rife, the association of men and cattle is less close, and the anopheles are anthropophilic [see HACKETT & MISSIROLI, *ante*, p. 569].

Four years ago the Commission instituted an inquiry into the value of the various unseparated mixtures of the cinchona alkaloids,

* Suspected to be carriers on epidemiological grounds only.

or quinetum. As the result of this enquiry they consider that the value of quinetum is well proved, and they recommend its use both on account of its therapeutic value and its cheapness in comparison with quinine.

The Committee of the League organized a vast experiment in a number of hospitals, with Quinoplasmine, which is the commercial name of the newer tablets of plasmoquine compound, each of which contains 0·01 gm. of plasmoquine and 0·3 gm. of quinine sulphate. They now recommend a daily dose of 4 tablets for a period of 10 to 21 days. With this treatment, 100 per cent. of definite cures have been obtained in quartan; only 4 per cent. of relapses have occurred in benign tertian; the crescents have disappeared in 5 or 6 days in subtertian.

W. F.

SOUTHERN MEDICAL JOURNAL. 1931. May. Vol. 24. No. 5. pp. 407–463. With 8 text figs. (1 map.) [31 refs.]—**Symposium on Malaria.** [20 papers.]

The papers in this symposium were read before the National Malaria Committee at Louisville, Kentucky, in November 1930.

Dr. HACKETT contributed a most interesting paper on the Control of Malaria in Italy (see pp. 122–128, above). Associations of landowners for financing and executing drainage schemes have existed in the Po Valley since the fourteenth century; over a hundred years ago, the grand dukes of Tuscany initiated a colossal project which will take yet another 25 years to complete. In all, the Italians have reclaimed more land from the waters than the Dutch. This land reclamation (bonification) has both hygienic and agricultural objectives. The most successful form of bonification is the filling of low land and lagoons by sedimentation. Units of 10,000 acres or more are bunded to the desired level, and the rivers are turned into them at flood. When the bunded area is full, the water is allowed to stand for 48 hours and is then led off over a spill-way to make room for a fresh charge. Such a process of warping, or filling on a large scale, by the sedimentation of natural waters is going on in the coastal area of Tuscany. In a good rainy season, a deposit of silt a foot deep can be obtained. The financing of bonification is made possible by the high value of well-drained agricultural land. The Government usually offers 75 per cent. of the total cost in fifty annual instalments, covered after the first decade by the taxes on increased production. The owners pay the remaining 25 per cent. The value of the land may rise from sixteen shillings to as high as £40 an acre after drainage. The soundness of these schemes depends upon the large agricultural population of the country which leads to an immediate intensive cultivation of drained areas and to a good market for produce. In countries with a sparse population, such a scheme would not be profitable. In northern Italy, where the relative absence of aestivo-autumnal malaria allows immigration and agriculture to begin at once on the newly drained land, the attention of *A. maculipennis* becomes directed to the numerous well-stabled animals, its association with man gradually ceases, and there is "anophelism without malaria." In some important cases, malaria has not been affected by drainage, the land has not been occupied and the project has been a failure; for example, the Tiber delta, though drained with pumps since 1889, remained highly malarious and 90

per cent. uncultivated for more than 30 years. Agriculture is impossible there, in the long dry season, without irrigation, and malaria was an insuperable barrier to colonization. *A. maculipennis* is a mosquito of the north temperate zone: in southern districts like the Tiber delta it seeks the coolest water, and breeds almost exclusively in the vegetation along the quiet edges of streams and drainage canals. This is why drainage, of the Panama type, though constantly attempted in Italy, has met with so little success. The failure of the delta scheme led to the adoption of State quinine in 1902. The State manufactures it, but the landowners have to pay for it and for the treatment of their labourers. Malaria is legally a notifiable, occupational disease in Italy. What has been the effect of State quinine? "We may sum up the situation very briefly by saying that the effect on severity has been marked and incontrovertible. The death-rate from malaria has rapidly descended, and cases of pernicious malaria are far less frequent now than before. The effect on incidence has been almost negligible."

Bonification is a slow process, and in recent years four other measures have been adopted: (1) *Concrete Channels.* These have been laid in the flat bottoms of the drainage canals. Such channels, which keep the water moving and are easily cleaned, with the aid of electric pumps and gambusia, have been sufficient to eliminate anopheline breeding from the area surrounding Ostia in the Tiber delta, and to abolish malaria; (2) *The introduction of Gambusia.* These little fish, introduced from the United States, multiply so astoundingly in Italy that standing waters become clogged with them to such an extent that the peasants complain that the cattle refuse to drink. In an area of about 8 square miles, in Istria, where nothing has been done but gambusia distribution, the spleen rate has gone down from 98 per cent. in 1924 to 10 per cent. in 1930, and a sanatorium which was formerly closed in the summer, because of malaria, is now kept open all the year round without a single case occurring among its 300 inmates. (3) *Larvicides.* There are many places where malaria is due to river-breeding anophelines, and here Paris green is employed. This is comparatively easy in Italy because the agricultural population lives in compact villages, and not scattered over the land. Paris greening of all unavoidable breeding places within 1½ miles of population centres is the standard practice in Italy. The local authorities carry this out, and the landowners bear the expense. (4) *Screening.* The Government of Rome, in 1928, made it obligatory for all householders of the Campagna to screen. Perhaps the most important advance was the "Law of Mussolini," passed in 1928, under which the reclamation of all fertile but uncultivated land must be begun within ten years.

Dr. W. H. Taliaferro read a paper on the mechanism of acquired immunity in avian malaria. When canaries are infected with *P. cathemerium*, an acute period, with a rapid increase of parasites, is followed (if the bird does not die) by a crisis during which nearly all the parasites suddenly disappear. About a week later, the bird appears to have recovered entirely, and parasites can no longer be found, but inoculation of large quantities of its blood into healthy birds shows that there is a latent infection. This period of latent infection may last for years and, while it lasts, the bird is immune to further infection. The author found that the inoculation of blood from such immune birds did not produce immunity in normal birds. He attributes the immunity to an increase in the number of phagocytic cells, and to a greatly increased rate of phagocytosis.

Dr. Eugene WHITMORE read a paper on plasmoquine in which he deprecates its use in blackwater fever. As he points out, the drug has no action on the malignant tertian schizonts, and the destruction of the gametocytes cannot influence the course of the disease.

W. F.

UNITED FRUIT COMPANY, BOSTON, MASS. NINETEENTH ANNUAL REPORT. MEDICAL DEPARTMENT. 1930. Section II. pp. 17–60. [**Papers on Malaria.**]

Dr. W. E. DEEKS, in his comments on the more important diseases occurring in the tropical divisions, states that, "in carrying out a blanket treatment in a heavily-infected district, we believe that the average adult individual should receive one tablet of plasmoquine compound and ten grains of quinine sulphate twice daily for a period of six days, . . . After an interval of two weeks . . . the six day period of treatment is repeated on all of the individuals who are then found positive . . . When repeated blood surveys prove that the malaria incidence has been reduced to less than 30 per cent., efforts should be centered on the identification and treatment of all individual infected cases." [One tablet contains 0·01 gram of plasmoquine and 0·125 gram of quinine.]

There has been a progressive improvement in the Cuban Divisions. A colonization scheme has resulted in a great increase in the permanent local population, including women and children. Homes have been established, schools provided, fruit and vegetable gardens made, and labour stabilized. The camps are regularly inspected and the sick are treated. A remarkable decrease, not only of malaria, but also of other diseases has been the result.

Drs. R. B. NUTTER and J. C. MCDANIEL of Honduras conclude "that attempts at mosquito control and intensive sanitary work alone will not reduce malaria to an extent which will justify the expenditures involved to effectually control mosquito breeding in the large areas of a banana division." They pin their faith on "systematically surveying all labour camps and treating all positive cases."

Dr. J. R. MALTSBERGER of the Chiriqui Land Company, a rapidly developed division on the Pacific coast of Panama, has carried out antilarval work by filling, draining, oiling, and Paris green, in addition to treatment with plasmoquine and quinine, and he considers "that further progress undoubtedly lies in anti-larval control administered with energy and foresight." Employees who refuse treatment have their pay stopped, and other recalcitrants are fined by the local magistrates. As a result of all these measures there has been a progressive decrease in malaria.

Dr. N. P. MACPHAIL of Guatemala says that "we have learned from experience that malaria cannot be eradicated from large tracts of tropical low-lying lands . . . with anti-mosquito measures alone. The treatment of the human carrier is a most important measure."

Drs. H. C. CLARK and L. H. DUNN, of the Gorgas Memorial Hospital, Panama, report an attempt to transfer monkey malaria to man [*ante*, p. 494]. Nothing resulted from their efforts to transfer the parasites either by mosquitoes or direct blood inoculation, and they conclude that "red spider-monkey malaria and the human benign species of malaria that it so closely resembles are not identical, and that the

monkey is not, therefore, a reservoir for human malaria." Two specimens of *A. tarsimaculatus* and one of *A. albimanus*, fed on monkeys, showed positive glands on dissection.

W. F.

LANE (Clayton). **Housing and Malaria. (A Critical Summary of the Literature dealing with this Subject.)**—League of Nations. Health Organisation. C.H. Malaria/169. L.o.N.P. III. Health. 1931. III. 6. 43 pp. With 4 figs. [8 pages of refs.] Geneva. [1s. 6d.; $0·40.]

The following is a summary of the author's conclusions :—

"The anopheles associated by experience in the transmission of malaria are in the main house-haunters and night-biters, circumstances which make it likely that the house is important in the transmission of malaria . . . . There are 'malarious houses' where inhabitant after inhabitant acquires malaria. These houses have certain characteristics, at least in Europe; they possess dark, dirty, and often damp portions. In parts of Europe and in indigenous dwellings in the tropics, a chimneyless fire is efficient in driving away anopheles . . . this condition in the past must widely have influenced the incidence of malaria in Europe; with the invention of the chimney it needed the further institution of ample lighting before houses again became distasteful to anopheles. . . . It is the case that infected anopheles may be particularly associated with certain houses, and the question has been considered as to whether these mosquitoes have an instinct to return to a pleasant hospice after they have left it to oviposit. Such evidence as exists for an instinct of this kind is questionable; the facts are perhaps explicable as showing a tropism towards food, and, if so, they emphasize the importance of abolishing breeding-places near houses . . . Certain species of anopheles leave the house where they have fed immediately after feeding, others remain in it for some hours. There is, however, at least in Europe, Northern America and Brazil, a condition of gonotropic dissociation, when the need to oviposit ceases, while the need to feed does not. Mosquitoes in this condition remain in the house where they have fed, and they are capable of becoming malarious and of transmitting malaria. . . . There is no satisfactory evidence that malaria-carrying anopheles prefer the blood of animals to that of man, or that airless animal houses near human habitations necessarily protect man from the bites of anopheles. . . . In the early days of the construction of the Panama Canal, the destruction of replete anopheles in houses was held with good reason . . . to have proved a most effective anti-malarial measure; . . . The value of the screening of houses was established once and for all by the experiment designed by Sir Patrick MANSON in the Roman Campagna to test his mosquito-malaria theory in 1900. Its success showed that, if anopheles were prevented from entering houses, malaria could be avoided, . . . it is in the house that malarial infection is essentially obtained; . . . Various lines of investigation press with cumulative force the conclusion that the house is a factor of primary importance in the acquisition and spread of malaria."

W. F.

WATSON (Malcolm). **The Lesson of Mian Mir.**—*Jl. Trop. Med. & Hyg.* 1931. July 1. Vol. 34. No. 13. pp. 183–189. With 1 map. [4 refs.]

Anti-malaria operations at Mian Mir, a great military cantonment in India, were begun in 1902 by S. P. JAMES, continued in 1903 by S. R.

CHRISTOPHERS, and afterwards by other officers of the Indian Medical Service. JAMES stated in the discussion which followed a paper on "the Control of Malaria" read by Dr. L. W. HACKETT before the Royal Society of Tropical Medicine and Hygiene (*ante*, p. 124), that the operations at Mian Mir showed that mosquito reduction in that locality was difficult and expensive, and that the discovery of the mosquito cycle of the malaria parasites had not put within our grasp a simple and cheap anti-malarial weapon. Sir Malcolm Watson denies emphatically that the experiment at Mian Mir proved anything of the kind, for several reasons: (1) *The men in charge of the work were inexperienced.* "It was, for India, new work. . . . Was it likely then that from a few months' experimental work, two young men, . . . should be in a position to say, not only the first but the last word for all time—on practical anti-mosquito work?" (2) *Money was wasted on useless projects, and wrong measures were adopted.* In 1902, 74 per cent. of the money was spent in bricking and plastering a single water-course, which JAMES himself said, afterwards, was constructed on an entirely wrong plan. Money was also wasted on such futile work as bailing out pools and digging mud out of the bottom of drains. There is plenty of evidence in JAMES's report that the larval control was quite inefficient. (3) *Insufficient oil was used.* It should have been applied thoroughly once a week, but during the whole malarial season of 1902, only Rs.350 was spent on "contingent expenses for kerosine oil, materials, etc.". In the report of the operations in 1903 there is no statement of expenditure, nor is it possible to ascertain the amount spent on the work done in 1904–1909. (4) *Absence of a proper zone of protection.* In order to protect dwellings it is necessary to remove them half-a-mile from breeding places, or to surround them with an area half-a-mile broad, artificially freed from larvae, but, in the operations of 1902, every point inside the treated area was within half-a-mile of a boundary, and therefore not a single house within it was efficiently protected from malaria. In 1903, things were not much better, for the protecting zone was three-quarters of a mile wide in some places, and much less than half a mile in others.

"The anti-malarial operations at Mian Mir were in fact badly designed, and inefficiently carried out. As evidence of the cost, ease or difficulty of conducting, or the value of practical anti-malarial work either there or elsewhere, the experiments were worthless."

W. F.

IYENGAR (M. O. T.). **Absence of Malaria in the Salt-Water Lake Basin.** —*Indian Jl. Med. Res.* 1931. July. Vol. 19. No. 1. pp. 163–174. With 1 map in text & 2 plates. [8 refs.]

On the eastern side of Calcutta is an extensive, inland, saline-water basin, ten miles long and three miles broad. There are many small villages, on islands in the basin, inhabited by fishermen. For many years, the unhealthiness of Calcutta has been ascribed to the proximity of this salt-water lake; but the author's survey has shown that the basin is a healthy area almost entirely free from malaria, the gross spleen rate being only 0·3 per cent. Anopheles are very prevalent, but are almost entirely limited to the single species *A. subpictus* Grassi (*rossii* Giles), and this "seems to prove conclusively that this mosquito is a non-carrier in nature." An interesting point in connexion with

this area is the total absence of *A. ludlowi* in spite of the conditions, such as brackish water containing Enteromorpha, being apparently very favourable; so far as can be judged there are no essential differences between the malarious fishponds of the coastal region of the Dutch East Indies, in which *A. ludlowi* breeds profusely, and the salt-water lake basin.

W F

SWELLENGREBEL (N. H.). **Report on Investigation into Malaria in the Union of South Africa, 1930-31.**—Union of South Africa. Department of Public Health. 45 pp. 1931. Pretoria. Govt. Printer. Also published in *Jl. Med. Assoc. South Africa.* 1931. July 11 & 25. Vol. 5. Nos. 13 & 14. pp. 409–424; 443–456.

It is important to distinguish between farm-malaria and estate-malaria. In *farm-malaria* the problem wholly concerns the white population, it scares away new settlers and injures those already there; fever among the natives may be widespread, but it does not affect the labour-supply, which is indigenous and tolerant. In *estate-malaria* the fever amongst the native labour force is the main problem, in so far as it is recruited from non-malarious parts of the Union. It affects the cultivation of valuable crops and so causes serious financial losses. Malaria occurs in two distinct epidemiological types. There are *endemic areas*, in most of which little is heard of malaria though they comprise some of the worst fever areas in the Union. The reason for this is that the inhabitants have learnt to deal with malaria by quinine, screening, and the like, in such a way as to render life bearable. Then there are *epidemic areas* where malaria occurs in outbursts, with long intervals of freedom, and causes great alarm and popular outcry.

Among the 20 species of South African anopheles there are only two, *A. costalis* and *A. funestus*, which are of importance as carriers of malaria. *A. costalis* breeds in muddy puddles, provided these are exposed to sunlight, contain no vegetation at all, and are continuously refreshed by rain water. If rain stops for more than 7 days, these breeding places disappear. *A. funestus* is a stream-breeder; its larvae are found in the grassy edges of the backwaters of quickly-running hill streams and rivers. *Costalis* malaria is dependent on local rains, but *funestus* malaria is not. The occurrence of an appreciable number of *costalis* means malaria. Not so with *funestus*; in some places, there are swarms of *funestus* with no malaria, because, in these areas, they do not go into the houses; in neighbouring districts, though the numbers of *funestus* are no greater, there is a great deal of fever because the mosquitoes enter the houses to bite the inhabitants. The cause of epidemic malaria in South Africa is fairly obvious. It is mainly due to an unwonted increase of *A. costalis*, owing to an extension of its breeding area in consequence of rainy periods succeeding each other at short intervals. This allows of a fairly reliable forecast, based on the rains, or, better still, on the development of *costalis*. The alleged disappearance of malaria from an area when it is settled and planted is not due to agricultural development, which, by irrigation, provides more facilities for mosquitoes; "the instances of so-called disappearance of the fever from highly malarious areas are explained by the Europeans gradually adjusting themselves to local conditions, in a country where there occur little or no changes in the native population

and where influx of settlers is scarce." The equilibrium established is unstable, and the introduction of natives, unaccustomed to the diseases from some non-malarious area, means not only disaster for them, but also for those already settled in the district.

Most large collections of water are unimportant as breeding places; neither *costalis* nor *funestus* breed in the papyrus swamps or in the "vleis"—the swampy areas at the bottoms of valleys. "But beware of touching these swamps, as it is the easiest thing in the world to render them dangerous . . . . The surest way to render even a large swamp dangerous is to drain it, and not to take care that the drainage furrows are either full or empty. Puddles. . . are almost sure to breed *costalis*." Touching the question of "clearing," or cutting down vegetation as an anti-malaria measure, because it harbours mosquitoes, there is not sufficient evidence that it does so, and the only places where we find many anopheles taking shelter are houses and clefts between moist rocks. In dry years, pools form in the beds of rivers and *costalis* breeds in them. In a rainy year, all these breeding places are washed away, but they reappear in the middle veld, farther from the rivers, as rain water puddles. In the low flat country, one is in an area of almost pure *costalis* malaria; in the foothills with their numerous streams is the region of *funestus* malaria; above 3,000 feet *funestus* disappears because it is too cold. The lower temperature does not affect *costalis*, and altitude to get away from malaria is not to be relied upon if *costalis* can establish itself: it is the absence of suitable puddles in the hills which prevents its breeding. "The source of infection for anopheles in an endemic area is the native population, and the place of infection the native hut . . . There are conditions rendering the prevention of malaria in the Transvaal an easier problem than in any other country I know of, and others which make it the most difficult I have ever seen. The former relate to the insect vector, the latter to the human carrier." The stream-breeder, *funestus*, is not very difficult to deal with, and *costalis*, by its almost complete dependence on local rains, is an ideal subject for malaria prevention by anti-larval control. The great difficulty is the population; the white settlers are scattered all over the country, living on isolated farms, far from medical assistance. Malaria in the native location is not a danger to the white man.

Prophylactic quininization "is the most hope-inspiring anti-malarial measure" in the Transvaal. The spleen rate of 39 children belonging to non-quinine-taking families was 77; the spleen rate of 52 children belonging to quinine-taking families was 52. In some areas, screening is widely practised, and though, as a rule, far from perfect, the screened houses contained only a tenth of the anophelines found in the unscreened, and the spleen-rate of the children was 50 per cent. less. In epidemic areas the inhabitants take few precautions, because the discontinuity of the disease gives insufficient incitement to the people to exert themselves. A great deal may be accomplished by the district surgeons seeking contact with the people and visiting them in their homes, not only instructing them how to treat themselves with quinine, and how to deal with anopheles breeding places near their farms, but also teaching them the importance of screening and of killing mosquitoes inside their houses. In many districts the diet is deficient in green vegetables and fruit; here, again, the district surgeon's advice is necessary. He should receive the help of district nurses of the health visitor type. "Additional training of the district surgeon and his influence being multiplied by district nurses is one of the most promising anti-malarial

measures in the Transvaal." Free quinine should be supplied only to those genuinely unable to pay for it; otherwise the public loses a sense of its value even to the extent of buying patent so-called remedies instead of using it.

An experiment in control should be undertaken in an endemic area where there is much malaria. Each farmer will have to do, or at least supervise, the work himself. The farmers have such wrong notions about dangerous breeding places that they need the most detailed advice, and this should be given by a specially trained sanitary inspector. They should be taught how to oil, or Paris-green, all collections of water, indicated on a sketch-map of their farm, once a week for the 4 months January to April. It is improbable that farmers in epidemic areas would adopt this scheme, but as the *costalis* breeding places (the only ones of importance in these areas) are limited to the rivers in non-epidemic years, the farmers should be advised to live at least half a mile from their banks. If the authorities wish to establish settlements of poor whites in highly malarious areas, the houses should be united into one closely-built village, on a site outside a *funestus* area. All the houses must be screened and there must be a sanitary inspector for each village, but the anti-larval work must be done by the villagers themselves. "No amount of endemic malaria need prevent European settlement, provided the necessary personal precautions are taken, viz., prophylactic quinine, screening, regular killing of mosquitoes inside the house, and segregation of natives."

Malaria on the estates in the sugar-belt of Natal is almost entirely due to *costalis*. All breeding places around the complex of European establishment and native compound (which should be close together for the purpose of supervision) should be dealt with by filling with sand or oiling. It is a very simple matter. Attention must be paid to the general principles of estate sanitation, and the Government should institute labour inspection to see that the necessary measures are carried out. All cases of illness lasting more than three days should be treated in hospital, so that accurate knowledge may be obtained of the diseases occurring on the estates, and proper records may be kept. The importation of natives from non-malarious areas into the sugar-belt is sometimes responsible for outbreaks. This danger can be avoided by employing labour recruited from intensely malarious districts where there is a high degree of tolerance. This tolerance is not immutable; bad food and bad sanitation can destroy it. Children from an area like this are dangerous to their surroundings, but the adults are not. Such malaria-tolerant labour should be employed on railway construction in fever areas, but not in regions free from malaria.

As regards the treatment of malaria among the native population: in highly endemic areas there is great tolerance of malaria, even the heavily infected children suffer comparatively little, and the mortality among them is low; the adults are healthy, suffer only occasionally from fever, and a very small dose of quinine will set them right again. These people are not eager or thankful for quinine, and they are better without it, because the process of immunization among them would be hindered by quininization. Further research is necessary in these endemic areas. In epidemic areas the natives are anxious to take quinine during an epidemic and the Natal system of native malaria assistants, who initiate treatment in the reserves during epidemics, should be continued.

As regards research and instruction, what is required is intensive

research continued for a long time at a field station for malaria control, established in a highly malarious district, so that the influence of seasonal changes may be estimated and entomological and epidemiological findings may be closely co-ordinated.

W. F.

SYMES (C. B.). **Report on Anophelines and Malaria in the Trans-Nzoia District.**—*Kenya & East African Med. Jl.* 1931. June & July. Vol. 8. Nos. 3 & 4. pp. 64–77, 108–121. With 3 charts. [4 refs.]

Kitale, the centre of the district which lies north of the Nzoia River, is situated 1° north of the equator, but as the altitude varies from 5,000 to over 8,000 feet, the temperature is pleasant and the district is one of the best settled in Kenya. There are many streams and the rainfall is about 45 inches. Until settlement was begun in 1919, the area was practically unoccupied owing to the warlike activities of the Masai. Larvae of the following anophelines, in order of numerical prevalence, were found during the present survey: *A. natalensis; christyi; mauritianus; costalis; funestus; squamosus; implexus; transvaalensis; nili; cinereus; pretoriensis; marshalli; maculipalpis; rhodesiensis; pharoensis.* Those most obviously associated with malaria were *A. costalis* and *A. funestus;* other species were too rarely found in houses to be of importance. Precipitin tests carried out on the stomach contents of captured adults, demonstrated the preference of these mosquitoes for human blood; out of 167 *A. costalis,* 137 contained human blood; out of 812 *A. funestus,* 499 contained human blood. In most of the huts, the mosquitoes have an equal opportunity of feeding on livestock. Sporozoites were found in 7 out of 90 *A. costalis,* and in 1 out of 15 *A. funestus.* The importance of distance from a breeding place is not the same on all farms; "on one farm it may be easier for adults to fly a distance of 600 yards than 100 yards on another because of the local vegetation and topography." The author believes that fewer Europeans would be infected if all the African staff were housed in one spot so that anopheles in only one area became infected; at present, native huts are scattered broadcast, and the unfortunate settler is surrounded by a ring of infection. Blackwater fever is most common about 2 months after the peak of subtertian malaria, when *P. malariae* is the most prevalent parasite; on these grounds the author suggests that "blackwater may be a manifestation of a superinfection of subtertian on quartan malaria (or vice versa) carried by *A. costalis* and *A. funestus* respectively."

W. F.

SERGENT (Edm.), SERGENT (Et.), PARROT (L.), FOLEY (H.), CATANEI (A.) & SENEVET (G.). Etudes épidémiologiques et prophylactiques du paludisme, 26e, 27e et 28e campagnes en Algérie en 1927, 1928 et 1929. [**Antimalarial Campaigns in Algeria, 1927-1929.**]—*Ann. Inst. Pasteur.* 1931. Apr. Vol. 46. No. 4. pp. 457–466.

The year 1927 saw the end of the steady decline of malaria in North Africa, and in 1928 it flared up after heavy rain. The amount of malaria in each of these years was predicted from the rainfall in the spring. In 1927, following a dry spring, there was little malaria, except

in a few spots where it had been wet ; in 1928, after a wet spring, there was a great deal. In places where prophylactic precautions were taken, there was less malaria than elsewhere. Energetic combined operations were undertaken in 1928, by the anti-malaria service, the district medical officers, the district engineers, and the school teachers. Quinine, 6 grains in a capsule daily for adults, and a smaller dose for children in a chocolate pill, was widely distributed ; anti-larval measures were undertaken, and the people were instructed by films and lectures. As a result there was less malaria in 1929 than was predicted on meteorological grounds, except in the untreated areas where the disease continued unabated. Since 1902, certain test areas have been selected in the most highly malarious parts of Algeria, and practically every known anti-malaria measure has been tested in these places. The experiments have shown that it is possible to reduce the splenic and parasitic indices to negligible proportions. The authors conclude that modern methods are efficacious, and accelerate the improvement which occurs much more slowly under natural development. The expense, however, is great, and the extent of success is limited by the money at command.

W. F.

FEDERATED MALAY STATES. **Annual Report of the Malaria Advisory Board for the Year 1930** [WILSON (Christopher J.), Chairman].—22 pp. With 4 folding sketch diagrams. 1931. Kuala Lumpur : Govt. Press.

The Chief Health Officer reported that there had been a slight increase in the number of deaths from all causes, as compared with the previous year, and that this increase was largely the result of malarial conditions. Special reference was made to outbreaks in certain places such as Port Swettenham, notorious in the past. An interesting item in the Report is a reference to drought as a cause of malaria : in one district " an epidemic of malaria . . . had been provoked by cessation of oiling, which had resulted from the fact that every drop of water was required for drinking and washing purposes."

W. F.

STEWART (A. D.). **Notes on a Visit to the Malay Peninsula.**—*Indian Med. Gaz.* 1931. Aug. Vol. 66. No. 8. pp. 457–465. With 3 text figs.

" Malaria is now a serious factor on many estates, and would still be everywhere were the conditions preventing it relaxed for even a year or two. It is a universal topic on every estate and dominates every other medical consideration." The earlier method of subsoil drainage has been modified by Dr. HUNTER of Singapore, with much success. His method is briefly as follows : The ravine is first cleared of trees and shrubs ; then three, deep, open drains are dug, one central and two lateral contour drains. These are left as earth drains for six months, and oiled if necessary. This lowers the subsoil water, and shows how deep it will be necessary to make the permanent central drain. This is then graded for treatment, commencing from below upwards. Dr. HUNTER prefers an open central drain, with concrete inverts, having

projecting studs to keep a space between the joints, in order to admit seepage water. The sides of the drain are grassed down to the inverts or, better, are revetted with flat earthware slabs. It is seldom necessary to pipe the whole of the contour drains ; in most of their length they are simply filled with stones. At points where seepage has been noted, a short length of subsoil drain is put in, with a branch running down to the central drain, in which no gradient above 1 in 30 is permitted. The author concludes that " the methods used in Malaya are narrowed in their scope by the limited number of carrier species that have to be dealt with. These methods even apart from the cost question would have a limited field of application in India."

W. F.

NIEUWENHUIS (A. W.). Die Entstehung der Polynesier und ihrer Kultur. Der Einfluss endemischer Malaria in Ozeanien. [**Influence of Endemic Malaria in Oceania.**]—*Acta Leidensia* (*Scholae Med. Tropicae*). 1930. Vol. 5. pp. 128–192.

In this lengthy monograph on the culture of the Polynesians the author quotes extensively from Professor Macmillan BROWN's book, " The Riddle of the Pacific," published in 1924. A quotation states that, " No masterful or imperial race ever came out of the tropics or ever will come out of them. Even in the warmer parts of the temperate zone the rulers of empires generally come from the colder north." Again, " The imperial ambitions that produced a Kamehameha and a Pomare in an environment that was marked by licentiousness and decay must have had its origin and heredity in some climate different from that of the tropics." The author is not inclined to accept these views. He contrasts the populations in malarial and non-malarial districts. He concludes that not only the physical qualities, but also the mental are much higher in populations living in non-malarial districts than in those living in endemic areas. In support of his conclusion that endemic malaria has played an important part in the deterioration of the culture of the people of Oceania he quotes the opinions expressed in the work by JONES, ROSS and ELLETT, " Malaria a neglected Factor in the History of Greece and Rome."

E. D. W. Greig.

RICE (E. Milford). **On the Economic Aspect of Malaria to Tea Estates in the Bengal-Dooars.**—*Jl. Trop. Med. & Hyg.* 1931. July 15. Vol. 34. No. 14. pp. 193–204. With 3 graphs in text.

The author estimates that on an estate of 1,600 acres, worth £140,800, malaria causes a loss of £4,555 a year, or more than 3 per cent. on the capital. This is on the basis that a cooly is worth £30 to the estate. He concludes that it would be justifiable to spend £900 per annum to reduce the incidence of malaria by 50 per cent. " Nothing is more evident than that at the present time very large amounts of money are being frittered away by estates in the Dooars for useless anti-malarial work, which may, in fact, be causing a very great increase in the infection rate," by cutting the jungle over streams and making them suitable breeding places for *A. maculatus* and *A. minimus*.

W. F.

MACDONALD (G.) & CHOWDHURY (K. L.). **Report on a Malaria Survey of the Tea Gardens in the Mariani Medical Association, Assam.**—*Records of the Malaria Survey of India.* 1931. Mar. Vol. 2. No. 1. pp. 111–156. [5 refs.]

This paper deals with malaria on eighteen estates controlled by seven tea companies which are grouped together for medical purposes under the Mariani Medical Association. The gardens lie near the foot of the Naga Hills. The dangerous carriers are *A. minimus* and *A. culicifacies*. The chief recommendations made by the authors as the result of their survey are that greater care should be taken in the selection of sites for cooly lines, and that the principal method of attack should be by making the water unsuitable for breeding by the growth of vegetation; where this has failed, or has not been done, Paris green should be used as a temporary measure. Paris green should also be used on swamps and pools where *A. culicifacies* breeds.

W. F.

MANSELL (R. A.). **A Discussion on Malaria in the Quetta-Pishin District.**—*Jl. Roy. Army Med. Corps.* 1931. June. Vol. 56. No. 6. pp. 401–409. With 5 charts & 1 sketch map.

Quetta is about 5,500 feet above sea level. The district consists of precipitous, barren, mountain ranges, enclosing alluvial valleys. The soil is light and porous; natural vegetation, except in the immediate vicinity of the few permanent waters, does not occur. The winter rains and snow provide water for the crops, but during the greater part of the year it is a sandy, dust-coloured country, burnt dry by the sun and blown bare by the wind. Water supplied for irrigation disappears into the ground with such amazing rapidity that cement water channels are only a handicap to the malaria worker. In Quetta cantonment the breeding places of mosquitoes are the bricked siphons carrying water under the roads, a few places near the main irrigation channel, and running taps in gardens. The features of the climate are high diurnal and annual variations of temperature, low rainfall, and low humidity. Malaria is endemic on the plateau, and occasionally becomes epidemic. There is not often much mosquito breeding in Quetta itself, but in the surrounding country, wherever there is a constant flow of water with marginal vegetation, mosquitoes breed freely from the middle of May to the middle of September. Many species of anopheles occur among which are *A. culicifacies*, *A. listoni*, *A. stephensi*, and *A. superpictus*. The author discusses the possibility of *A. stephensi* breeding in the "karezes." The karez is started from a point in a valley where water outcrops; and, from this point an ever-deepening channel is dug backwards towards the source of water in the hills. When the channel becomes too deep it is converted into a tunnel following the direction of the waterflow. At intervals, shafts are sunk down to the tunnel, which is only large enough to permit the passage of one man and may be many miles in length. The shafts in Quetta, of a karez which runs through the cantonment, are nearly 80 feet deep. The karezes are constructed with the most primitive implements by people who follow this work as a hereditary occupation, and they form the main sources of water for large tracts of country.

W. F.

WILKINSON (Sydney Arthur) & STUBBS (W. D.), assisted by Vaikunthrai Manilal DESAI & Kunwar BEHARILAL. **A Malaria Survey of Ajmer City 1930.**—pp. viii+30. With 5 text figs., 1 folding map, 1 folding chart & 1 folding plan. 1931. Bombay: The Times of India Press.

Ajmer, the second city of Rajputana, lies on a plateau 1,500 feet above sea-level, and is surrounded by hills. It has a population of 113,000, about half of whom are employed on the Bengal, Baroda, and Central Indian Railway. The spleen index shows that it is an area of moderate endemicity, but outbreaks of malaria occur after heavy rain. This survey was made by the authors in their spare time. They found that the important anopheles was *A. stephensi*, and that its breeding places were the waste water from the public stand pipes, garden sumps, tubs, pots and pans, and also the pools left near the edges of a couple of artificial lakes when these were drying in the hot weather. They concluded that the malaria of Ajmer was man-made and easily preventable. The application of municipal regulations already in existence would reduce malaria considerably, and if even the small amount at present spent on anti-malarial work by different bodies were scientifically utilized there would be a rapid decrease of the disease.

W. F.

SUN (T. C. Y.) & YOUNG (M. P.). **A Report on 167 Cases of Malaria treated in Elizabeth Blake Hospital during 1930.**—*China Med. Jl.* 1931. Mar. Vol. 45. No. 3. pp. 258–265. With 2 diagrams & 2 charts.

The results of examining the blood of out-patients and in-patients showed that statistics based on the examination of a hospital population are not always a true index of the relative prevalence of the three different kinds of malaria. The figures for in-patients were 70 per cent. subtertian; 24 per cent. benign tertian; 4 per cent. mixed; and only 2 per cent. quartan. Among the out-patients no less than 32 per cent. were quartan cases, the explanation being that the milder cases do not go into hospital.

W. F.

YOFÉ (Hillel). Le paludisme en Palestine. [**Malaria in Palestine.**]—*Rev. Prat. Malad. des Pays Chauds.* 1930. Dec. Year 9. Vol. 10. No. 12. pp. 552–563. [15 refs.]

Palestine is well suited for major drainage operations because it runs down in steep slopes to the sea on one side, and, on the other, to the valley of the Jordan which ends in the Dead Sea, 400 metres below sea level. The most important anopheles are *A. elutus*, which breeds in stagnant pools full of weeds, and *A. sergenti* which breeds in slowly running water. Much has been accomplished by improved cultivation and organized colonization; in this respect the author pays tribute to Baron Edmond de Rothschild, who inaugurated the scheme of Jewish colonization in 1888. Drainage, intensive agriculture, and anti-larval measures, have been almost everywhere successful. For example, Petah-Tikvah, in Judea, was formerly a poverty-stricken village surrounded by a collection of Bedouin tents; since a Jewish colony

was founded there it has become the centre of the orange plantations, with a population of about ten thousand, and malaria, which was once prevalent, now occurs only sporadically. In Jerusalem itself, where storage tanks are one of the principal causes of malaria, the government is introducing a water supply. The author advocates the free distribution of quinine in all places where more radical measures of malaria prevention cannot be undertaken or have not been completed.

W. F.

Wolski (M.). Zur Klinik und Epidemiologie der Malaria nach den Angaben der Malariainstitutionen der Stadt Samara. (Mitgeteilt auf dem 3. Wolgakongress für Malaria in Samara.) [**Symptomatology and Epidemiology of Malaria as met with in the Malarial Institutions of Samara.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1931. Aug. Vol. 35. No. 8. pp. 471–482. [18 refs.] [Malaria Station, Samara-Satoust Rly., Samara.]

In this investigation the results are recorded of a study of 16,765 malarial cases during the years 1923, 1924, and 1925. The great majority of malarial infections were benign and malignant tertian. In 1923 the predominating form was benign tertian; quartan was only observed in the early stages. In 1925 there was an increase of malignant tertian. The number of mixed infections increased with each year. There appears to be a territorial distribution of the forms of malaria, but no connexion between age periods and the different forms. The spleen and Ross index are highest in quartan and mixed infections. These indices reach their maxima during childhood and gradually diminish as age advances. The spleen index diminishes as the city is approached, the lowest being in the city itself. The liver was found enlarged in the majority, most frequently in quartan infection, less frequently in benign tertian and least in malignant tertian. There is a close association between liver and spleen enlargement. Each form of malaria has a distinct curve of fresh infections with a peak at a definite period of the year.

E. D. W. Greig.

Rybinsky (S. B.). **Further Observations on the Malarial Curve in Kiev.**—*Zent. f. Bakt.* I. Abt. Orig. 1931. Aug. 11. Vol. 121. No. 7/8. pp. 409–413. With 2 text figs. [14 refs.] [Bact. Inst., Kiev, Ukraine.]

Malaria was imported into Kiev in 1920 by famished refugees from the valley of the Volga where the disease was malignant. At first, the malaria curve showed two peaks, one in spring and one in autumn, but, under the influence of the cold summers of Kiev and the improved conditions of the people, the malarial plasmodium began to lose its virulence; the autumn rise slowly disappeared, the disease became less prevalent and, at the same time, it assumed a milder form. The spring rise, which is due to the occurrence of cases with long latent periods, infected in the autumn, is a manifestation of a weakening of the malarial virus under the influence of a cold climate. The treatment in the spring, before the mosquito season, of persons previously ill with malaria has been successful in reducing the disease.

W F.

JANCSÓ (Nicolas) & D'ENGEL (Rodolphe). Observations sur les réviviscences du paludisme aux environs de la ville de Kolozsvar, pendant une période de cinquante ans. [**Fifty Years' Observations on Recrudescent Malaria in the Neighbourhood of Kolozsvár.**]—*Riv. di Malariologia.* 1930. Nov.–Dec. Vol. 9. No. 6. pp. 725–733. With 1 text fig. [Internal Clinic, Univ., Szeged, Hungary.]

These epidemiological observations were made at the François-Joseph University during the period 1873 to 1919, and concern a circumscribed area in the west of the Transylvanian basin. This area includes the town of Kolozsvar, with 60,000 inhabitants, and 116 villages with a population of 120,000. During the 50 years of observation, there have been three waves of malaria: the first appeared in 1870 and reached its crest in 1877; the second appeared in 1890 and culminated in 1897; the third wave did not appear until 1916. During the intervals between these waves of endemic malaria, cases of the disease are very rare. Why is this? No drainage, regulation of watercourses or bonification has been carried out. There is no evidence that meteorological conditions play any part in it. Anopheles (*maculatus* and *bifurcatus*) are as numerous and as easily infected in the malaria-free years as in those when the disease is prevalent. Experiment has shown that the fall of the wave is not due to an immunity which it has provoked in the population; moreover, the proportion of the inhabitants which becomes infected is too small to make this a factor. No quinine prophylaxis or screening is carried out. The author concludes that malaria occurs in wave-like oscillations, like the waves of pandemic influenza, uninfluenced by external conditions and due to unknown causes.*

W. F.

BARBIERI (Antonio). El paludismo y los factores meteorológicos y heliofisicos en la República Argentina. Su epidemiologia. [**Epidemiology of Malaria and Meteorological Factors in the Argentine.**]—*Prensa Méd. Argentina.* 1931. Apr. 30. Vol. 17. No. 33. pp. 1616–1628. With 4 graphs. [23 refs.]

The author discourses on certain meteorological factors in the Argentine and on the prevalence of malaria in various districts and from a correlation of these is of the opinion that future epidemics are predictable. The chief endemic zones are the Provinces of Jujuy, Salta, Tucuman, Catamarca, La Rioja and Santiago del Estero, where there are well watered, shaded valleys, exuberant vegetation, a mean annual temperature of 20–21° C. and abundant rain. The important vectors of malaria are *A. pseudopunctipennis*, and to a less degree *A. argyrotarsis* and *A. albimanus*.

During the last 50 years, those of large epidemics have been 1878, 1889, 1901–2, 1913–14, 1919, 1924, 1930–31, that is "a typical cycle" of 11–12 years, and "less typical" of 5–6 years. In Paraná and Uruguay, the cycles were closely similar, 1858, 1868, 1878–79, 1888–89, 1901, 1912, 1924, 1929, and 1931.

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* CELLI pointed out that since the middle of last century severe malarial epidemics had occurred in the Roman Campagna with approximately 10-yearly frequency. Mild outbreaks alternated with the severe epidemics [see this *Bulletin*, Vol. 24, pp. 411 et seq.].

In a graph are shown the correlations between temperature, barometric pressure and solar radiation and the author notes that at intervals of 11·1 years there has been exceptionally heavy rainfall, reduction of solar radiation, absence of sun-spots, and that this period coincides with the revolutions of Jupiter (11·86 terrestrial years), and he concludes that taking into account the atypical cycles of approximately five years he can predict epidemic exacerbations of malaria in subtropical South America, between 20° and 40° south latitude every 5–6 years starting from 1930.

H. H. S.

GERBER (C. W.). **Summary of Malaria Control Work in Dona Ana County, New Mexico.**—*Southwestern Med.* 1931. Aug. Vol. 15. No. 8. pp. 370–375.

For 30 years or more, malaria was unknown in the county, but in 1915 large irrigation works were commenced and, in 1918, it became necessary to construct drainage canals, because the land was becoming water-logged. The drainage canals have been continually extended and they have been allowed to become choked with weeds. The population has increased by 63 per cent. since 1923, and, owing to the fluctuating demand for cotton-field workers, people are continually coming and going. With these changing conditions, malaria appeared in 1925, and has become a serious problem. *A. pseudopunctipennis* breeds in the sunlight, in the centre of the drainage canals, and *A. maculipennis* breeds near the shaded edges. "The use of a combination of methods such as dust diluted Paris green, oil mixtures, oil and Paris green mixtures, Paris green and wet sand, the propagation and distribution of top minnows, the instruction of the inhabitants . . . . adequate treatment . . . . has resulted to a comparatively high degree in reducing the Anopheles mosquito production, the number of local transmissions, and the total number of cases of malaria."

W. F.

FERRELL (John A.). **Challenge of Malaria in the South.**—*Amer. Jl. Public Health.* 1931. Apr. Vol. 21. No. 4. pp. 355–374. With 8 text figs. [1 ref.] [Internat. Health Division, Rockefeller Foundation, New York.]

The first table in the paper gives the death rates from malaria for the Registration Area of the United States from 1910 to 1929 and for the 9 southern states of the Union for 1918–29. The author points out that the figures for the Registration Area are not comparable throughout the period as only 9 out of the 16 states classed as malarious were included within the area and in 1910 only California. Table IA gives details of certain states, the death rates of which during 1919–21 exceeded 1 per 10,000. Table II gives the deaths and population of Virginia and of 10 south-western counties of Virginia. For the state as a whole the deaths recorded vary from 81 in 1918 to 13 in 1928. Table III records deaths and populations for Kentucky, Tennessee, Mississippi, Virginia, North Carolina and South Carolina for the years 1918–29. Each seems

to show a declining rate from 1921 to 1927, and some a tendency to increase in the following two years. Table IV gives details for subdivisions of North Carolina. Table V records the experiences, 1918–29, (for two States, 1915–29) of Arkansas, Texas, Missouri, Alabama, Florida, Louisiana, Georgia. Table VA gives details for the counties of Florida, Alabama and Georgia. The trends seem similar to those noted for the states included in Table III. Tables VI and VII give the death rates, for whites and negroes separately, from malaria for subdivisions of Mississippi, 1914–28. Table VIII records the death rates (1919–29 in three cities, 1925–29 in the fourth) of four southern cities. The author thinks that the factors making for the general recession of malaria have not yet been convincingly evaluated. The increase of mortality from malaria in Florida and Alabama in 1929 to a value higher than recorded in 1921–22 and the smaller increases in other affected states make it " clear that malaria is still a menace to the health and working efficiency of the people of the South and that the measures directed against it were inadequate for holding the gains through wet years."

M. Greenwood.

KORTEWEG (P. C.). **What is the Normal Type of Fever in Primary Cases of Benign Tertian Malaria? Is it Tertian or Quotidian?**—*Jl. Trop. Med. & Hyg.* 1931. Aug. 1. Vol. 34. No. 15. p. 251.

The author found that the normal type of fever was tertian [see also p. 591, *ante*], whereas S. P. JAMES on the contrary noted that it was quotidian and suggested " a possible explanation of the different results observed in the two countries may be that unavoidably Dr. Korteweg's work was done upon patients some of whom had previously suffered from an unrecognized malaria attack." Dr. Korteweg holds that the differences between the types of fever observed in England and Holland must be due to some other cause ; his mental patients came from all parts of the Netherlands, where, with the exception of a few small foci, malaria is endemic in only one of the eleven provinces.

W. F.

ALESSANDRINI (Mario). Sulla trasmissibilità del *Plasmodium falciparum* dalla madre al feto. (**On the Transmissibility of** " *Plasmodium falciparum* " **from Mother to Infant.**)—*Riv. di Malariologia.* 1931. Mar.–Apr. Vol. 10. No. 2. pp. 197–201. With 3 figs. on 1 plate. [8 refs.] English summary (4 lines) pp. 266–267.

The natural placental filter usually prevents the passage of malarial parasites when present in the mother's blood from passing to the foetus, but small infarcts, capillary lesions and such like may result in the obstacle being overcome. In the author's opinion the parasites are only present in the foetal blood (umbilical cord) when they are very numerous in the placenta, and even then are exceedingly scarce in the former. When practising in Leccese, a locality in which the population is " nearly 100 per cent. malarious," the author took the opportunity

of examining the peripheral blood of mothers in parturition, of the placenta, and of the umbilical cord. He gives brief notes of 10 patients, all malarious ; in two there were parasites at the time in the peripheral blood, but in nine there were none in the placenta or cord. The tenth was a woman suffering from bilious remittent fever ; her blood showed *P. falciparum*, the placental blood was heavily invaded, 90 per cent. of the red cells being parasitized, the umbilical blood showed the parasite in the same stage as those in the mother, but exceedingly few, only 12 or so, were found in a search of several hours. He did not find any nucleated red cells invaded. [The " leucocytic formula " of the foetal blood is given as megaloblasts 10, erythroblasts 38, polymorphonuclears 34, lymphocytes 16, large mononuclears and transitionals 2 per cent. !]

H. H. S.

i. Chantriot. Syphilis, paludisme et sérologie. [**Syphilis, Malaria and Serology.**]—*Rev. Prat. Malad. des Pays Chauds.* 1931. June. Year 10. Vol. 11. No. 6. pp. 249–56, 259–61. [16 refs.]

ii. Trabaud (J.). Les réveils accidentels de l'infection palustre. [**Incidents which arouse Latent Malaria.**]—*Ibid.* pp. 262–4, 267–8.

iii. Hakim (Assad). Quelques formes nerveuses et mentales de l'infection palustre. [**Some Nervous and Mental Diseases due to Malaria.**]—*Ibid.* pp. 269–272.

i. Captain Chantriot has found that an attack of malaria has the same beneficial effect upon visceral or latent syphilis as it has upon syphilis of the nervous system. He has seen many cases which had previously resisted specific treatment, respond to it at once after an attack of malaria, and many in which an obstinately positive Wassermann reaction has become negative. Shock produced in other ways, for example by the inoculation of foreign proteins such as a vaccine or horse serum, or by auto-haemotherapy, can act in the same way.

ii. Professor Trabaud contributes a useful paper on the accidents which may awaken a latent malaria ; infectious diseases such as influenza and typhoid ; therapeutic inoculations, such as injections of arsenobenzol and colloidal salts ; protein shock, after vaccine or serum treatment ; exposure to heat and cold ; poisons and trauma, such as alcoholism, fractures and childbirth.

iii. Dr. Hakim gives an account of three cases seen in the Asylum of Avicenne at Damascus. One with bilateral paralysis of the circumflex nerves ; one with mental confusion and somnambulism ; one with Parkinsonian symptoms. In each case he considers that malaria was the cause.

W. F.

Bonnin (Henri). L'avenir des paludéens rapatriés en pays non impaludé. Le paludisme de guerre en 1931. [**The Prospects of Malaria Patients on Return to a Malaria-free Country. War Malaria in 1931.**]—*Gaz. hebd. Sci. Méd. de Bordeaux.* 1931. Apr. 19. Vol. 52. No. 16. pp. 245–253. [10 refs.]

There are two groups of malaria patients in France : those who became infected in Macedonia during the war, and those who have

contracted their infections in the French colonies. The course of the disease appears to be quite different in these two groups. Malaria in the old colonial disappears in a year or two after his return to France, but this is not the case with the war veteran in whom the disease still appears to flourish. At a congress of old soldiers held at Caen in 1930, it was agreed that all who had been infected with malaria during the war ought to receive a pension of 10 per cent. Subtertian infections generally disappear within a year of return to a non-infected country, and benign tertian within two or three; quartan sometimes lasts longer, but there was hardly any quartan malaria in Macedonia. Cases of long latent infection have been recorded: RUGE, 9, 10, 13 years; SCHILLING, 8 years; PLEHN, 6 and 13 years; BLANCHARD, 6 years; NOCHT, 8 years; but these are all exceptional cases and were recorded as such. The law, the Press and many doctors, believe in these long-lasting war-infections as much as the patients do themselves. Every kind of illness and accident which happens to an old soldier who was infected during the war is put down to malaria: diabetes, aortic disease, hemiplegia and epilepsy are all attributed to malaria. The author has recently seen a man given 60 per cent. retired pay for a polyneuritis supposed to be due to malaria contracted more than ten years ago in the war, although there were no signs or symptoms of the disease. He has examined numbers of these war cases and has found neither the malaria parasites nor the enlarged spleen which would be present if they were really suffering from the disease.

W. F.

VIALATTE (Ch.) & SAINTE-MARIE (P. E. Flye). Autour du "mystère" de la fièvre quarte (remarques épidémiologiques). [**The Mystery of Quartan Fever.**]—*Bull. Soc. Path. Exot.* 1931. Apr. 15. Vol. 24. No. 4. pp. 280–282. [2 refs.]

Quartan malaria constituted 6·5 per cent. of all cases of malaria admitted to the Cocard native hospital, in Morocco, during the three years 1928–30. The percentage of quartan among the troops during the same period was only 0·34 per cent. The authors attribute the different incidence in the two groups to the prophylactic quinine taken by the soldiers. [They say that quinine acts more rapidly and completely upon the parasites of quartan than upon the other forms. This is contrary to the experience of many observers; the relative prevalence of quartan in the natives may be due to its chronicity.]

W. F.

TOPORKOW (F. M.). [**Epidemiology of Quartan Malaria.**]—*Trop. Med. & Vet.* Moscow. 1930. Vol. 8. No. 8/9. pp. 4–7. [In Russian.]

The author has studied the statistical material on the incidence of quartan malaria in Russia, and arrives at the following conclusions: The prevalent idea that quartan malaria occurs only in certain foci is based on isolated examinations. The examination of the records for five successive years shows that this type of malaria is distributed in all malarious localities. There appears to be a certain periodicity in its occurrence; in the majority of localities quartan malaria is prevalent during the autumn and winter months.

C. A. Hoare.

MACDONALD (G.). **The Mechanism of Infection with Malaria in Children living under Endemic and Hyperendemic Conditions.**—*Indian Jl. Med. Res.* 1931. Apr. Vol. 18. No. 4. pp. 1347–1372. With 7 graphs. [10 refs.]

This paper is based on the results of nine series of blood and spleen examinations made during a survey in Assam (see Macdonald and CHOWDHURY above, p. 987) and on work done in Sierra Leone. Repeated examinations over a period of 6 days showed that only about 60 per cent. of infections are discovered by the examination of a single thick film. In endemic areas such as Assam, it may be assumed that all children with enlarged spleens are infected, yet the parasite rate among these enlarged spleen cases is only 55 per cent.; therefore 45 per cent. of the infections are missed. To obtain an idea of the true infection rate in such a district the general parasite rate should be multiplied by a factor based on this error. This true infection rate and the spleen rate show a relationship to each other which cannot be explained except on the ground that only some 50 per cent. of infections produce enlarged spleens. Analysis of the Assam figures showed that (1) The parasite rate reaches its height in the first two years of life, except in the less malarious lines. (2) The numerical frequency of parasites in the blood decreases rapidly after the first two years. (3) The spleen rate reaches a maximum in children aged 3 to 6, and thereafter declines. (4) A relative immunity to malaria is gradually acquired by the children.

W. F.

GARNHAM (P. C. C.). **Observations on *Plasmodium falciparum* with Special Reference to the Production of Crescents.**—*Kenya & East African Med. Jl.* 1931. Apr. Vol. 8. No. 1. pp. 2–21. With 2 charts & 3 plates. [23 refs.]

This paper describes observations on some 200 cases of malaria in the native hospital at Nairobi. Warrington YORKE found that spleen puncture films differed little from peripheral blood films made at the same time (*ante*, p. 1). The author ascribes this to the examination of splenic blood drawn off with a syringe. He obtained spleen pulp by inserting a wide bore needle into the spleen between the lower ribs, closing the butt with the finger before withdrawal, and blowing the contents onto a slide. When this technique was employed schizonts were found in nearly every case. Crescents appear suddenly in the peripheral blood on the eleventh day of the disease, increase to a maximum, and then decline. The average duration of the crescent wave is 14 days, but crescents may persist for 66 or even 128 days, when there are frequent relapses. GREEN's conclusion that quinine increased the output of crescents, on the ground that 50 per cent. of his cases had crescents on admission, but 64 per cent. after 7 days' treatment (see this *Bulletin*, 1929, Vol. 26, p. 931) was evidently reached in ignorance of this law (see also AMIES, above p. 597). Quinine in the early stages tends to inhibit crescent production. Crescents are most plentiful in new cases and relapses; less so in chronic, latent, and recrudescent malaria. In 15 post-mortems, crescents were found in the spleen and bone marrow of about half the cases: none was found in the brain, liver, skin or large intestine. A characteristic feature of

the developing crescent is a dense ring of protoplasm on the outside. The spleen and bone-marrow are the nurseries of the crescents. "There is some slight evidence of parthenogenesis of the macrogametocyte."

W. F.

ZIEMANN (Hans). Ueber die etwaigen Beziehungen gewisser cumarinhaltiger Pflanzen zur Epidemiologie der Malaria. [**Relation of Certain Plants containing Coumarin to the Epidemiology of Malaria.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1931. July. Vol. 35. No. 7. pp. 410–420. [2 refs.] [Path. Museum, Univ., Berlin.]

The author quotes the views of WILLCOCKS [this *Bulletin*, Vol. 25, p. 557], D'HERELLE and KRYSTO on the absence of malaria from areas in which wild clover is prevalent and the presence of the disease in parts of the country where there is no wild clover. He considers that these are interesting and important observations and has compiled and published in this paper a long list of plants containing coumarin. Profs. BRAUN, GILG, Drs. LIEBISCH and MELCHIOR collaborated with him in this investigation.

E. D. W. Greig.

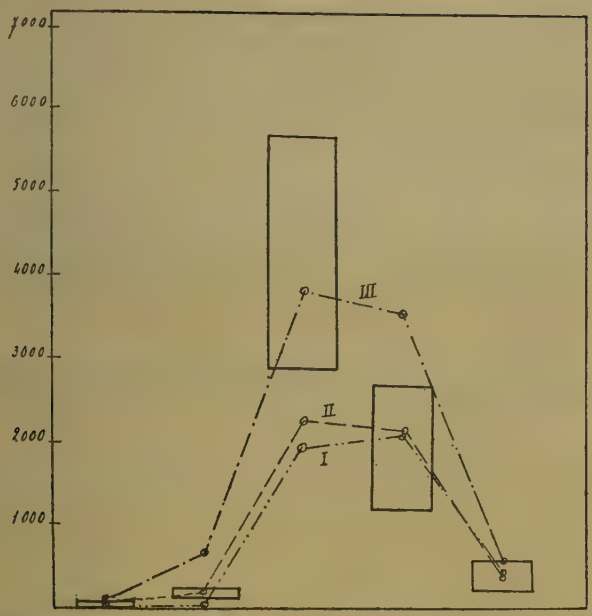
GILL (C. A.). **The "Control" of Malaria with Special Reference to Treatment.**—*Indian Med. Gaz.* 1931. June. Vol. 66. No. 6. pp. 333–335.

In this lecture to the Punjab Branch of the B.M.A. Col. Gill emphasizes the point that the prevention and treatment of disease are so closely connected that they cannot be separated. As the treatment of ankylostomiasis prevents the occurrence of fresh cases, so does the treatment of malaria prevent more malaria, for example on CLEMESHA's tea estates (*ante*, p. 576).

W. F.

MOCHKOVSKI (Ch.). Critérium leucocytaire de la persistance de la malaria. [**The Leucocyte Test in Persistent Malaria.**]—*Bull. Soc. Path. Exot.* 1931. July 8. Vol. 24. No. 7. pp. 544–551. With 4 text figs. [4 refs.]

What are the criteria of a definite cure and how can latent malaria be detected? The author claims that his "leucocytic profile" answers these questions. The profile is drawn as follows: An absolute count of the white corpuscles and also a relative count of the different forms is made, and from these the absolute number of each group of cells in a cubic millimeter is calculated. A graph is drawn showing the numbers of basophiles, eosinophiles, neutrophiles, lymphocytes, and monocytes, in the order given, at points equidistant along the base-line. These points are joined to form the curve which is called the leucocytic profile. In a normal blood, the neutrophiles form a sharp peak; but in cases of uncured malaria the curve is flattened, because there is a reduction in the absolute number of these cells. In the figure given below, points which fall within the rectangles lie inside the normal limits; for example the number of neutrophiles in curve III is normal, but the number of eosinophiles is not. When the cure of malaria is established, the neutrophile point on the profile falls into the neutrophile rectangle, and this is a sound proof that there is no latent infection. There is no



Illustrating method of plotting the "leucocytic profile" for three cases (see text of Mochkovski's summary).

			Basophiles.	Eosinophiles.	Neutrophiles.	Lymphocytes.	Monocytes.	Total.
Benign tertian	I	Per cent.	0·3	1·0	43	44	11	—
		abs.	10	50	1900	2000	500	4500
,, ,,	II	Per cent.	1·2	4·8	44	42	8	—
		abs.	60	240	2200	2100	400	5000
Lymphocytic reaction with eosinophilia of unknown cause	III	Per cent.	1	8·5	43	41	6·5	—
		abs.	100	800	4000	3800	600	9300

[Reproduced from the *Bulletin de la Société Pathologie Exotique.*]

real increase of monocytes in malaria, but only a relative rise, due to the diminution of neutrophiles.

W. F.

JORGE (Ricardo). Epidémies nautiques de malaria à forme typhoidique, pouvant faire suspecter la fièvre jaune. [**Ship Epidemics of Typhoidal Malaria, suggesting Yellow Fever.**]—*Bull. Office Internat. d'Hyg. Publique.* 1931. Apr. Vol. 23. No. 4. pp. 682–692. With 1 plate, 3 charts & 1 map. [2 refs.]

In the autumn of 1930 there were severe epidemics of malaria on board cargo boats arriving at Lisbon from ports on the west coast of

Africa (Senegal, the Gambia, Portuguese Guinea). The symptoms in some of the cases resembled typhoid ; in others, the rapidly fatal issue, the visceral lesions, and the generalized icterus suggested the diagnosis of yellow fever. Details are given of an outbreak on board a German steamer, which arrived at Lisbon with 10 of her crew of 21 down with fever. The symptoms were sudden onset of high fever, severe headache, diarrhoea, abdominal pain and, in one, abundant epistaxis. Four died ; in one, there was generalized icterus : the liver, kidneys and myocardium were yellow and fatty, there was follicular enteritis and gastritis. [There is no mention of black vomit.] The characteristic albuminuria of yellow fever was not present, and examination of the blood and organs demonstrated the presence of *P. falciparum* in large numbers.

W. F.

SURBEK (K. E.). **On Renal Reactions and Nephritis in the Course of Malarial Infections.**—*Riv. di Malariologia.* 1931. Mar.–Apr. Vol. 10. No. 2. pp. 194–196. [6 refs.]

The author recognizes four types :—(1) *Febrile Albuminuria* due to pyrexia ; light and transitory, and occurring in 8–10 per cent. of cases. (2) *Renal Reaction* (*sensu stricto*) with more albumen than the first, and with casts. This form is most common in subtertian. It soon clears up if it is properly treated, but if not it goes on to (3) *Acute Nephritis.* This also responds to quinine, if not left too long. (4) *Quartana-Nephrosis infantum ac adolescentum,* which is a subacute parenchymatous nephritis, particularly associated with untreated quartan malaria in children and adolescents, and frequently accompanied by ascites. It responds to quinine, but there is a tendency to chronicity and relapse [see also GIGLIOLI, this *Bulletin,* Vol. 27, p. 508].

W. F.

JANCSÓ (N.) & D'ENGEL (Rodolphe). Sur la néphrite dans le paludisme. (**Nephritis in Malaria.**)—*Riv. di Malariologia.* 1931. Jan.–Feb. Vol. 10. No. 1. pp. 86–92. [1 ref.] English summary p. 158. [Internal Clinic, Univ. Szeged, Hungary.]

Nephritis is more common in some outbreaks than in others, and occurs more often in malignant tertian than in the other forms of malaria. It usually appears soon after the onset of the disease, and is of the acute haemorrhagic-type. Quinine acts as a specific. In some chronic cases of benign tertian and quartan, there is a chronic nephritis with cardiac hypertrophy ; similar cases occur sometimes in malignant tertian, but they are less frequent than the acute form of nephritis. In a wave of malaria which occurred during the years 1898–1901 the author found nephritis in 1·4 per cent. of 2,190 cases.

W. F.

NICOLAU (C. T.) & GINGOLD (N.). Modifications hématologiques pendant les accès de malaria. [**Blood Changes during Malarial Paroxysms.**]—*Bull. et Mém. Soc. Méd. Hôpit. de Bucarest.* 1931. Feb. Vol. 13. No. 2. pp. 19–20. [Filantropia Hosp., & Inst. of Hyg. & Public Health, Bucharest.]

The examinations were made just before, during, and at the end of an attack. The neutrophiles attain their maximum when the fever is

highest. The eosinophiles and monocytes diminish, or disappear, with the rise of temperature, and increase with its fall. The lymphocytes diminish with the fever and increase when the attack is at an end. The destruction of the red cells is greatest at the moment the temperature begins to rise. The number of corpuscles destroyed is 3–8 thousand.

W. F.

MANCA (Serafino). Sindrome appendicolare di natura malarica. (**Appendicular Syndrome of Malarial Origin.**)—*Riv. di Malariologia.* 1931. Jan.–Feb. Vol. 10. No. 1. pp. 93–98. [9 refs.] English summary p. 158. [Roy. Med. Clinic, Rome.]

The author calls to mind cases in which, on the one hand, patients in malarial districts have shown symptoms of acute appendicitis, but whose appendices when removed appeared in all respects normal, and, on the other, those who have exhibited no symptoms but whose appendicular vessels were thrombosed with malarial pigment.

He relates the case of a woman of 32, who had previously suffered from malaria, showing the typical signs of acute appendicitis—intense pain, at first generalized in the abdomen, settling in the right iliac fossa, a very tender McBurney's point, fever, nausea, vomiting, and characteristic decubitus with flexion of the right thigh. On the third day, these symptoms abated temporarily but returned in less than an hour. The spleen was enlarged. A surgeon was called in and a blood smear taken prior to operation. Numerous subtertian rings were found [the leucocyte count is not stated] and quinine given, 1 gm. by mouth and 1 gm. by injection. The pain was much relieved by the next day and two days later the fever had disappeared and the patient was comfortable. The quinine was continued in smaller doses, 1·5 gm. daily, after the third day and recovery was rapid. [The possibility of appendicular colic occurring in a malarial patient, apart from actual causal connexion, does not seem to have been considered, beyond the statement that "there was no peri-appendicular obstruction."]

H. H. S.

DECOURT (Philippe). Récidive de paludisme à neuf ans de distance. Transfusion du sang. Transmission du paludisme au donneur. Danger des transfusions de sang aux colonies. [**A Malarial Relapse after an Interval of Nine Years. Transmission to the Donor during Transfusion. The Danger of Transfusion in the Colonies.**]—*Rev. Méd. et Hyg. Trop.* 1931. Jan.-Feb. Vol. 23. No. 1. pp. 32–37. [1 ref.]

A man who had suffered from malaria in the colonies but who had lived near Paris for nine years, free from attacks and from possibility of re-infection, was admitted to hospital with a subcutaneous abscess. Some days after it was opened he had an attack of malaria. The author comments on the extreme rarity of such a long interval between infection and relapse. Before the diagnosis was made, the patient was transfused with blood, and the donor became infected with malaria. The author concludes that in cases where the receiver is suffering from a blood infection it is better to use citrated blood rather than direct arm-to-arm transfusion. The danger of transmitting infection is particularly great in the colonies, and the recipient should be treated with quinine. The donor should be treated as well, if he has schizonts in his blood. The author considers that if the donor has an old-standing malarial infection, it is useless to give him quinine, because this will not destroy the gametes which may

develop into schizonts after inoculation into the recipient. [But there is no cogent evidence that such a development takes place.] The type of parasite which infected the patients is not stated.

W. F.

DOROLLE. Paludisme grave au cours d'un traitement malariathérapique de la paralysie générale. [**Severe Malaria during a Course of Malaria Therapy in General Paralysis.**]—*Bull. Soc. Méd. Chirurg. Indochine.* 1931. Mar. Vol. 9. No. 3. pp. 153–154. [1 ref.]

A patient was inoculated subcutaneously with 5 cc. of blood from a naturally infected donor, in whose blood only *P. vivax* had been found. The patient developed malaria ten days later and nearly died. All three species of malaria parasites were found in his blood, and it is probable that the donor was infected with *P. falciparum* and *P. malariae*, in addition to *P. vivax*, though the former were too scanty to be detected by microscopic examination.

W. F.

SCHADOW (H.). Impfmalaria als Ursache kongenitaler Malaria. [**Congenital Malaria caused by Inoculation Malaria.**]—*Muench. Med. Woch.* 1931. June 5. Vol. 78. No. 23. pp. 947–948. [26 refs.] [Children's Clinic, Univ., Hamburg-Eppendorf.]

A female during pregnancy received a course of malarial therapy for general paralysis. After six rigors labour commenced and she gave birth to a living child. She had received one dose, 0·25 gm. of quinine, to modify the fever. Three weeks after birth the infant began to get attacks of fever and examination of the blood showed the presence of *P. vivax*. It became profoundly anaemic. At the sixth week of life the spleen was enlarged and extended to a hand's breadth below the costal margin. Blood transfusion was carried out and quinine administered. It was discharged from the Clinic at the seventh month of life in good condition although the spleen was still distinctly enlarged. Examined five months later the spleen was still palpable, but the child was otherwise healthy. No malarial parasites could be found in the blood even after administration of adrenalin.

E. D. W. Greig.

JANKELSON (I. R.). **Transmission of Malaria through Injection of Whole Blood.**—*Jl. Amer. Med. Assoc.* 1931. July 18. Vol. 97. No. 3. p. 177. [City Hosp., Boston.]

Several ounces of an Italian's blood were inoculated into his anaemic infant, which soon afterwards developed quartan malaria. Quartan parasites were also found in the blood of the father. He had no clinical symptoms of malaria, but admitted that about once in 12 or 24 months he had a chill which lasted for an hour and was followed by perspiration. He had suffered from malaria as a child, but he had lived in Boston for the last 20 years. The author concludes that "a man may be a carrier of the plasmodium for almost 40 years."

W. F.

SNIJDERS (E. P.). De behandeling der verschillende vormen van malaria. [**The Treatment of the Different Forms of Malaria.**]—Reprinted from *Aanwinsten op diagnostisch en therapeutisch gebied.* [1931.] pp. 443–484. [Refs. in footnotes.]

Clinically a fever of intermittent character recurring daily with shivering occurring at the same time of day must raise the suspicion of

malaria, but need not necessarily be malaria. Liver abscess, tubercle, otitis media, a septic focus, pyelitis, cystitis and lymphogranulomatosis are mentioned as some examples of non-malarial causation. One symptom which always accompanies the malaria attack is a marked urobilinuria.

The answer to the question whether quinine acts directly or indirectly upon the malaria parasite is not yet satisfactory, but the author maintains that the process of cure is essentially one of body resistance and not to be compared with *in vitro* tests of the death of the parasite. Quinine is not, therefore, a *therapia sterilisans magna*, capable of destroying the malaria parasite so to speak in the human test tube. Of all the medicaments, other than quinine, which may be used plasmochin is the most noteworthy. Its dosage has to be carefully adjusted, especially in ill-fed and enfeebled patients; a cyanosis may make its appearance due to methaemoglobinaemia and this should be watched for, not so much by a diminution of blood haemoglobin as by an increased urobilinuria. A very usual type of dosage is one of 10 mgm. plasmochin together with 250–300 mgm. quinine four times a day for 10 days. The administration of quinine, as the mainstay of malaria treatment, is specially considered. Its proper absorption can be followed by the addition to 5 cc. albumin-free urine of the reagent—pot. mercury iodide 10; glacial acetic acid 5; distilled water 100. A turbidity develops which disappears with heating and reappears on cooling. A choice of the many preparations of quinine presents a difficulty and it is well to know their varying alkaloidal content. Rate of absorption must also be taken into account. Dosage is regulated to some extent by weight, 1 gm. of hydrochloride for a 60-kgm. weight and 1·5 gm., but not more than this, for a 90-kgm. weight. A course of treatment at 200 mgm. 5 times a day for 5 or at most 10 days should be sufficient. After this the relapse should be watched for and the same treatment renewed. In this way 1, 2, or 3 series of treatments should suffice and this method is much better than a prolonged course. In cases of malaria where oral administration of quinine is impossible then, but only then, must injection be employed and this may be intramuscular or intravenous. Painful infiltrations, necroses, abscesses and lastly tetanus are to be avoided by strict attention to asepsis. Such a solution as quinine hydrochloride 3; antipyrin 2; water to 10 cc. fractionally sterilized at 80–90° C. gives no infiltration in a dose of 1·1 or 2·2 cc. along with 5 times the amount of sterile normal salt solution as diluent.

The quinine prophylaxis of malaria, admittedly a controversial subject, is shortly considered. It seems necessary to use what are really therapeutic doses such as 2/3–1 gm. daily for at least 10 to 14 days after infection or during an epidemic.

W. F. Harvey.

WILLOUGHBY (Hugh) & ASLETT (Edward). **The Treatment of Malaria and Blackwater Fever, with Notes on the Pre-Blackwater State.**—*Jl. Roy. Nav. Med. Serv.* 1931. Apr. Vol. 17. No. 2. pp. 95–103. [Hosp. for Trop. Diseases, London.]

"The *optimum maximum* oral dosage of quinine is 10 grains given *thrice* daily in B.T. and quartan infections, and 10 grains given *twice*

daily in M.T. cases. Any excess is likely to lead to toxic symptoms, especially in weakly individuals, and is waste of a valuable drug." As a general rule B.T. and quartan need three months' treatment. Plasmoquine compound is advised where there is intolerance of quinine, in pre- and post-blackwater fever cases, and in pregnant women. Four seven-day courses of 2 tablets twice a day are recommended, with four-day intervals, followed by a fifth course of one tablet three times a day. Each tablet contains 0·01 gram plasmoquine and 0·125 gram quinine. The patient should remain in bed for the first two courses. Glucose should be taken daily, and if toxic symptoms appear the dose of plasmoquine should be decreased. "Individuals who show signs of cyanosis are nearly always completely cured." [The daily dose of quinine, 12 grains, is not big enough for all cases of M.T. The quantity of plasmoquine seems unnecessarily large. Few malaria patients can be kept in bed for a fortnight.] The authors believe that a "pre-blackwater state" is readily recognizable; they consider the following points to constitute a pre-blackwater syndrome. All or some of the following signs and symptoms may be exhibited: (*a*) persistent headache; (*b*) dirty furred tongue; (*c*) icterus, especially noticeable in the conjunctivae; (*d*) nausea and vomiting; (*e*) loin pain, most marked over the kidneys; (*f*) enlarged tender spleen; (*g*) enlarged tender liver; (*h*) the presence of albumen, bile salts and bile pigments in the urine; (*i*) diarrhoea or sometimes constipation. [The "pre-blackwater state" is evidently far more common than actual blackwater.]

W. F.

VEDDER (E. B.) & MASEN (J. M.). **The Determination of Quinine in the Blood as a Guide to the Treatment of Malaria.**—*Amer. Jl. Trop. Med.* 1931. May. Vol. 11. No. 3. pp. 217–229. With 1 text fig. [5 refs.]

"The methods reported for the determination of quinine in the blood, not only make a scientific treatment of malaria possible, but open a door to a new line of experimental work on the toxicity of quinine to the various forms of the different malaria parasites." Patients suffering from benign tertian malaria were given 10 grains of quinine sulphate in capsules at 9 a.m. and 1 p.m. [Apparently irrespective of weight.] The quinine in the blood was estimated in samples collected half an hour, 1½ hours, and 2½ hours later. In a number of patients a very considerable concentration of quinine was reached within half an hour after administration. There is apparently no excuse for intramuscular injections, and intravenous injections should be used only in cases of coma where the drug cannot be given by the mouth. A single dose of 10 grains never produces the maximum obtainable concentration in the blood; in almost every case higher concentrations were obtained in the afternoon after the second dose of 10 grains. Some individuals must have an unusually efficient mechanism for either the excretion or destruction of quinine, for, after a dose of 10 grains one patient may have only 2 mgm. per litre in the blood and another 5 mgm. "This is quite probably the reason why, on a standard treatment, one individual will be cured, and another will relapse." The following methods were employed to estimate the amount of quinine in the blood:—

METHOD 1.

Preparation of Reagents and Standards.

"1. A 10 per cent. solution of silico-tungstic acid in distilled water.

"2. A 0·5 per cent. normal solution of hydrochloric acid.

"3. Quinine standards. A stock solution is prepared containing 200 mgm. anhydrous quinine in 1,000 cc. of 0·5 N HCl. 1 cubic centimeter of this solution contains 0·2 mgm.

"From this four standard solutions are prepared all dilutions being made with 0·5 N HCl.

"1. Five-tenths cubic centimeter stock solution is diluted to 100 cc. Five cubic centimeters of this solution contain 0·005 or 1 mgm. per liter.

"2. One cubic centimeter stock solution diluted to 100 cc. Five cubic centimeters contain 0·01 mgm., 2 mgm. per liter.

"3. Two cubic centimeters stock solution to 100 cc. Five cubic centimeters contain 0·02 or 4 mgm. per liter.

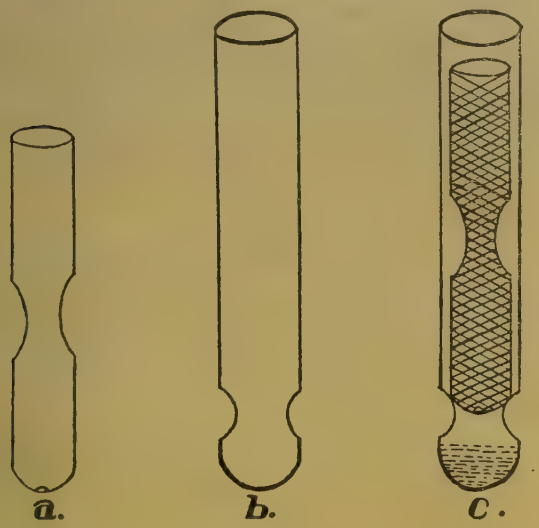


Fig. 1.

Extraction apparatus for determining the concentration of quinine in the blood.

[Reproduced from the *American Journal of Tropical Medicine.*]

"4. Three cubic centimeters stock solution to 100 cc. Five cubic centimeters contain 0·03 or 6 mgm. per liter.

"These standards cover routine procedure enabling estimations of quinine in the blood from 1 to 8 mgm. per liter. Higher standards may be prepared as required. These solutions deteriorate when exposed to light and must be kept in brown glass bottles. If protected from the light they are permanent for at least one year.

"*Technique.* A small circle of absorbent filter paper cut from a thick Whatman extraction thimble is placed in the bottom of tube "a" of the extraction apparatus, (Fig. 1) which may be made in any laboratory. This tube is then packed with long fiber, acid-washed asbestos to within about 2 cm. of the top. The asbestos must not be packed too tightly, otherwise the ether will not percolate through the blood properly. Neither must the

packing be too loose or the blood will not be properly absorbed on the asbestos and some will escape to the bottom of the tube into the ether. The constriction is placed in tube "a" in order to prevent blood from flowing down the walls of the tube thus escaping absorption by the asbestos.

"Five cubic centimeters of oxalated blood is pipetted on to the asbestos and a small pledget of cotton placed in the mouth of the tube, which is then inserted in tube "b." Ether is poured through the top of tube "a" until at least 5 cc. has percolated into the outer tube "b." The extractor is connected to a reflux condenser and is immersed in a warm water bath up to the level of the ether in tube "b." The extraction is then allowed to proceed for two hours. When extraction is complete, the inner tube is removed, and the outer tube containing the ether extract is placed in a boiling brine bath and evaporated to dryness. Five cubic centimeters of 0·5 N HCl is added and the tube returned to the brine bath for two minutes to facilitate solution of the quinine. The solution is filtered while hot, through a No. 42 Whatman filter, and the filtrate allowed to cool to room temperature.

"Three standards are prepared equivalent to 2, 4, and 6 mgm. per liter by measuring 5 cc. of the proper standard solutions into small test tubes. To each of the standards is added 0·1 cc. of the 10 per cent. solution of silico-tungstic acid, and to 3 cc. of the unknown is added 0·06 cc. of the same solution. Standards and unknown are then immersed together in a boiling water bath for five minutes, after which they are removed and cooled rapidly in running water. The unknown is then matched in the nephelometer against the nearest approximate standard."

Method 2.

Preparation of Solutions and Reagents.

"1. *Gum arabic.* Two grams of pure gum arabic (U.S.P. is suitable) is shaken with 100 cc. of distilled water until completely dispersed. The suspension in a Florence flask is immersed in a boiling brine bath for one hour to destroy reducing enzymes present in the gum, and is then filtered while still hot. On cooling the gum is ready for use, and will keep indefinitely if stoppered and kept in a refrigerator. It keeps about two months in the laboratory.

"2. *A stock standard* is prepared by dissolving 100 mgm. of pure anhydrous quinine in 500 cc. of 2 N sulphuric acid saturated with zinc sulphate. The working standards are prepared by diluting the stock solution 1 : 100, 2 : 100, 3 : 100 etc., with 2 N sulphuric acid-saturated zinc sulphate solution. Five cubic centimetres of these dilutions contain 0·01, 0·02, and 0·03 mgm. quinine respectively, or 2, 4, and 6 mgm. per liter. These standards must be protected from the light, by brown glass bottles. As the sensitivity of the test is affected by the concentration of the zinc sulphate, the zinc sulphate-sulphuric acid solution for the preparation of the standards should be the same as that used for dissolving the quinine extracted from the blood.

"3. *Two* N *sulphuric acid-zinc sulphate solution.* The purest crystalline salt, $ZnSO_4 \cdot 7H_2O$, should be used. The 2 N sulphuric acid should be saturated with the salt at temperatures somewhat below those encountered in the ordinary room temperature, as otherwise trouble will be experienced on account of the zinc sulphate crystallizing out as soon as the room temperature drops. Thus, if the minimum laboratory temperature is 20° C. the solution should be saturated at a temperature of 15 to 17° C., and should then be filtered.

"4. *Potassium bismuthous iodide reagent.* This is a reagent commonly used in the detection of alkaloids, and there are a number of formulas for its preparation. The one outlined gave the best results. Place 4·68 grams of bismuth oxide in 80 cc. concentrated HCl, and add water to 300 cc. Then dissolve 20 grams potassium iodide in water and dilute to 700 cc.

"*Technique.* Extraction of the blood is performed as described in Method 1. As soon as extraction is complete and the ether is evaporated, 5 cc. of 2 N sulphuric acid saturated with zinc sulphate is added, and the tube immersed in the boiling brine bath for three minutes in order to bring the quinine into solution. The solution while hot, is filtered through a No. 42 Whatman filter, and, after having cooled to room temperature, an aliquot of the filtrate, 3 cc., is measured into a small test tube. In three similar test tubes measure 5 cc. of each of the three quinine standard solutions. Immerse both standards and unknown in a cold water bath (20 to 25° C.) for five minutes in order to bring them to the same temperature. Then add to the standard 0·1 cc. and to the unknown 0·06 cc. of the gum arabic solution, followed by the same amounts of the potassium bismuthous iodide reagent (0·1 cc. to the standard and 0·06 cc. to the unknown). Mix and compare immediately in the colorimeter, with the standard set at 10 mm. The comparisons must be made within two minutes from the time that the solutions are mixed, as the color changes on standing. The calculation is made in the usual manner as described under Method 1.

"As in most colorimetric procedures, the closer the strength of the standard approximates that of the unknown, the more accurate will be the colorimetric matching. However for practical purposes it is sufficient to prepare the three standards just described and comparing the unknown with the nearest standard."

W. F.

MANSON-BAHR (Philip). **Quinine Therapy in Malaria.**—*Lancet.* 1931. Apr. 18. pp. 843–846.

The author believes that in the primary attack it is possible to extirpate the infection by prompt and energetic quinine treatment, but that in each subsequent relapse the parasites are more resistant. (JAMES, see above, p. 568, does not share this belief.) The removal of a patient to a temperate climate increases his bodily resistance, and shortens the life of the parasite. In a temperate climate, the life of the benign tertian is about three years, the quartan perhaps longer, and the subtertian only about one year. An agreeable way in which to give quinine in solution is as an effervescing mixture with citric acid grains 10, to which are added ammonium carbonate grains 4, and potassium carbonate grains 20. It is a great mistake, the author considers, to stop the quinine soon after the attack ; it should be continued, in benign tertian cases, for six weeks. Children should be given one-twentieth of the adult dose for each year of age. Intravenous injections of the bihydrochloride, 10 grains in 10 cc. of distilled water, are recommended in pernicious subtertian cases, and it is advised that 5 minims of a 1 : 1000 solution of adrenalin should be given at the same time in order to counteract the lowering of blood pressure produced by the quinine. Dr. Manson-Bahr thinks that great caution should be observed in giving quinine to anaemic patients with severe toxic subtertian malaria, because he is afraid of blackwater fever. He recommends a daily dose of no more than 5 grains to begin with, and its gradual increase. [Many tropical practitioners believe that the danger of producing blackwater is far less than the danger from the malaria parasites, and that, in such cases, the parasites ought to be destroyed without a moment's delay.]

W. F.

BLANC (Georges) & CAMINOPETROS (J.). De l'emploi de l'hydrate de quinine et d'hydrolactate de quinine par injections intraveineuses et sous cutanées dans le traitement du paludisme. [**Quinine Base and Quinine Lactate by Intravenous and Subcutaneous Injection.**]—*Rev. Prat. Malad. des Pays Chauds.* 1931. June. Year 10. Vol. 11. No. 6. pp. 275–8, 280–82. With 3 text figs. [3 refs.] [Pasteur Inst., Athens.]

To give quinine by the mouth is rather a shot in the dark, because one cannot be sure that the patient swallows it. [A drop of Mayer's reagent added to the urine will settle the question.] Most salts of quinine are likely to cause necrosis when injected ; therefore the authors tried quinine base. First they used " collobiase ", which gave good results but did not contain enough quinine. Subsequently they were able to obtain a solution containing 1 gram of quinine base in 5 cc. of water. [They do not say how this was done.] In order to obtain a more speedy action lactate of quinine was mixed with the quinine base [amount not stated]. After a number of trials they conclude that the solution is absolutely innocuous, and produces neither general nor local reaction when it is inoculated.

W. F.

HUGHES (T. A.). **The Effect of Intravenous Injections of Quinine on the Electrocardiogram in Man.**—*Indian Jl. Med. Res.* 1931. July. Vol. 19. No. 1. pp. 113–119. With 5 text figs. [4 refs.]

Quinine is a circulatory depressant and its administration by the intravenous route is not devoid of danger. Nine patients received 6 grains dissolved in 10 cc. of saline, and three 10 grains dissolved in 20 cc. of saline. The following are the author's conclusions :—

" The changes produced in lead II of the electrocardiogram by the intravenous injection of quinine in anti-malarial doses were studied in 12 patients. The most constant effect was a reduction in height or abolition of the T wave. Other effects observed were tachycardia, increase in height and width of the P wave, shortening of the P-R interval, reduction in height of the R wave and increase of the S wave. In one individual the P-R interval was reduced to 0·04 sec. Collapse, which was promptly relieved by adrenalin, occupied [? occurred] in two cases, in one of whom there was inversion of the T wave in leads II and III in the control curves. Symptoms were severe in this patient and were accompanied by marked electrocardiographic changes including depression of the R-T segment."

W. F.

SIDARI (Carlo). Il chinino nella malaria in gravidanza. [**The Action of Quinine in Malaria in Pregnancy.**]—*Riv. Sanitaria Siciliana.* 1931. May 1. Vol. 19. No. 9. pp. 674, 677–678.

The author discusses the action of quinine and states that in small, divided doses it affects the uterine muscle directly, but in large doses it has a sedative effect on that viscus while lowering the temperature and destroying the plasmodium. Moreover, it is but rarely that quinine initiates uterine contractions ; also, he states, statistics show that malaria has a very detrimental effect on pregnancy, " in 10 per cent. of cases it causes abortion, in 40 per cent. premature birth, in 20 per cent. death of the foetus " [the source whence these figures are obtained

is not given]. Hence, he is strongly in favour of giving quinine to pregnant women who are suffering from malaria, maintaining that it cures the malaria, and prophylactically prevents premature expulsion of the foetus.

H. H. S.

MANIFOLD (J. A.). **Report on a Trial of Plasmoquine and Quinine in the Treatment of Benign Tertian Malaria.**—*Jl. Roy. Army Med. Corps.* 1931. May & June. Vol. 56. Nos. 5 & 6. pp. 321–338; 410–423. [22 refs.]

The standard treatment adopted was: one tablet of 0·02 grams of plasmoquine together with 10 grains of quinine every morning and evening. This was given daily for 21 days, and in malignant tertian cases the course was followed by 0·04 grams of plasmoquine daily for 5 days. The treatment was entrusted to a number of medical officers in different parts of India, with instructions that a medical officer, or an assistant surgeon, should always be present when the dose was administered. The patients consisted of British and Indian soldiers, and 3,187 of them completed the 21 days' course. The following are the main conclusions:—

(1) The treatment may be given to all classes of British soldiers in India, irrespective of their physique. The same holds good for the great majority of Indian sepoys and followers, but in a few, about 0·1 per cent., it is possible that an attack of blackwater fever may be precipitated. "As in both British and Indian cases there appears to be a small percentage with a definite idiosyncrasy to the toxic action of plasmoquine, patients will require to be kept under observation, and should be excused duty during the three weeks' treatment." (2) As regards the prevention of relapses, "there is no doubt that it is *most* efficacious and a great advance on the ordinary quinine treatment." [Possibly, better supervision in giving the drugs was a factor.] The period of observation varied from 3 to 6 months. (3) It is definitely unsafe to issue plasmoquine to patients not under medical supervision. Good results might be met with in ninety-nine cases, and disaster in the hundredth. There were two deaths and two serious cases of methaemoglobinuria among the Indians.

Case 1 had benign tertian rings in his blood on the first day of treatment: on the 4th day, there were tertian gametocytes and crescents: on the 6th day, there were rigors and vomiting, with a temperature of 102·4° F., and plasmoquine was stopped: on the 7th day, there was marked jaundice, bile-coloured urine, severe abdominal pain and vomiting; the man died suddenly and unexpectedly in the afternoon. *Case* 2 had benign tertian rings in his blood when treatment was begun. On the 4th day, his temperature, which was normal on the 2nd and 3rd days, rose to 101·4°, no abdominal discomfort, no albumen in the urine: on the 5th day, temperature 102° F., passing dark coloured urine, seriously ill, plasmoquine stopped: 6th day, jaundice marked, bilious vomiting, cyanosis, methaemoglobinuria: 9th day, cyanosed, still passing port wine-coloured urine: 10th day died, after being unconscious for 48 hours. Post-mortem, fatty degeneration of liver and kidneys; renal tubules filled with amorphous debris. *Case* 3, had benign tertian rings. On the 5th day of treatment, his temperature, which had been normal for three days, went up to 104° F., there was marked jaundice, bilious vomiting, intense abdominal pain, and dark-coloured urine containing tube casts and blood corpuscles. He gradually recovered. *Case* 4. Benign tertian: jaundice appeared on the

4th day and plasmoquine was stopped : 5th day blood and bile in urine. He recovered quickly.

The author makes the following comments on these cases :—Case 1—" As a post-mortem was refused, the cause of death must remain uncertain. Neither the officer commanding the hospital concerned nor the medical specialist of the district was of opinion that death could be attributed to the plasmoquine." Case 2—" appears to have presented all the symptoms of a typical attack of blackwater fever." Case 3—" was similar in all respects also to a case of blackwater fever." Case 4—" appears to have been very much milder in type."

W. F.

KLIGLER (I. J.) & MER (G.). **Studies on Malaria VII.—Relapse Rate after Quinine-Plasmoquine Treatment.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1931. Aug. 8. Vol. 25. No. 2. pp. 121–127. [3 refs.] [Hyg. Dept., Hebrew Univ., Jerusalem.]

Forty-seven heavily infected children in the untreated Bedouin village of Melaha were treated as follows for 5 days :—18 were given quinine-plasmoquine mixture ; 17 were given quinine ; 12 were given no drugs. Their blood was examined weekly for 7 weeks, with the following results : there were 2 relapses in 17 cases [? 18] in the plasmoquine group ; 7 relapses in 17 cases in the quinine group ; 12 relapses in 12 untreated control cases. In the village of Almenieh, where treatment had been carried out during the previous year, the results were less favourable ; for out of 21 children treated with quinine-plasmoquine 12 relapsed. The treatment was therefore repeated at the first village, Melaha, with the result that there were 19 relapses in 53 cases. The authors conclude that though chinoplasmine [quinine-plasmoquine mixture] gives a lower relapse rate than quinine alone, the results of treatment in a group previously treated are not as favourable as in a virgin group : " partial treatment for gametocyte suppression . . . . apparently results in the selection of drug resistant strains." [The evidence given here cannot be accepted as conclusive.]

W. F.

DE MELLO (Froilano), BRÁS DE SÁ (L. J.) & D'ABREU (Mariano). Contribution à l'étude du traitement du paludisme par la plasmoquine.—[**Treatment of Malaria by Plasmoquine.**]—*Arquivos da Escola Méd.-Cirurg. de Nova Goa.* Ser. A. 1931. No. 6. pp. 715–753.

The authors confirm the general findings of others in the therapeutic use of plasmoquine. The patients treated by them were free from relapses if they stayed in a malaria-free locality. Seventeen per cent. of those who remained in the place where they contracted fever relapsed after treatment, but it was impossible to exclude the possibility of re-infection.

W. F

DE BUEN (Sadi). Nuevas notas sobre la plasmoquina compuesta en el tratamiento de la terciana benigna. [**Further Notes on the Treatment of Benign Tertian Malaria with Plasmoquine Compound.**]—*Medicina Países Cálidos.* Madrid. 1931. May. Vol. 4. No. 3. pp. 214–216. [1 ref.] [Antimalaria Inst., Navalmoral de la Mata, Cáceres, Spain.]

The author refers to his previous records (see this *Bulletin*, Vol. 26, p. 374) and gives an account of 97 children, from 1 to 15 years of age,

suffering from benign tertian malaria, treated between June and December 1929, for 3 weeks, the first and third weeks with plasmoquine co., the second with quinine only. Eleven of these (11·3 per cent.) relapsed in the first four months of 1930. Of 49 patients between 15 and 60 years similarly treated none relapsed. For children, therefore, it would appear that three weeks' treatment is inadequate ; the author recorded that when treatment was kept up for a month (in his previous trials) there were no relapses.

H. H. S.

DRENOWSKY (Angel K.). Die Malariabekämpfung mit Chinoplasmin in den Malariadörfern im Kreise Burgas während des Versuchsjahres 1930. [**Chinoplasmin as an Antimalarial Measure in Malarial Villages in Bulgaria during 1930.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1931. July. Vol. 35. No. 7. pp. 420–424.

As he was not satisfied with the treatment of acute malaria with plasmochin compound, in 1927 he increased the quantity of quinine and gave 1½ gm. and only 0·02 gm. plasmochin daily. He approves of Sinton's method, namely, for 10–20 days continuously daily doses of 1·2 gm. quinine and 0·04 gm. plasmochin. During 1930 tablets of chinoplasmin [each tablet consists of quinine sulphate 0·3 gm. (gr. 4½) and plasmochin 0·01 gm. (gr. ⅙)] were substituted. The tablets were efficacious and well tolerated ; although the quantity of plasmochin administered per day was small, in adults at most 0·04 gm. per day, the gametocytes disappeared from the blood in 5–6 days. Results were observed in 111 malarial cases. The investigation is still in progress and further results will be communicated in due course.

E. D. W. Greig.

MORISHITA (Kaoru). **Studies in the Treatment of Malaria. I. General Introduction and Routine Methods.**—*Taiwan Igakkai Zasshi* (*Jl. Med. Assoc. Formosa*). 1931. Jan. Vol. 30. No. 1 (310). [In Japanese. [9 refs.] English summary pp. 8–10.]

ODA (Teibun), MORISHITA (Kaoru) & NAMIKAWA (Hiroshi). **II. Experimental Treatment with Plasmoquine (I Report) Effect of the Short Course Treatment in Various Forms of Malaria.**—*Ibid.* [In Japanese. [1 page of refs.] English summary pp. 10–12.]

——, —— & ——. **III. The Therapeutic Effect of Saiko, an Indigenous Drug in the Orient, upon the Malarial Infections.**—*Ibid.* [In Japanese. With 8 charts. English summary pp. 12–13.] [Govt. Research Inst., Formosa.]

Drugs are tested on cases of chronic malaria among Formosan Chinese at the Institute for Experimental Malaria Therapy, established at Taihoku in 1929. There is hardly any possibility of re-infection there, but as a precaution the wards are screened. The patients are kept under observation for eight weeks after the cessation of treatment, and thick films are frequently examined. The term " cure " signifies no parasitic relapse during this period. When 0·04 to 0·06 gram of plasmoquine were given to patients with benign tertian and quartan, the relapse rate was 12·5 per cent. in the first, and nil in the second. [The numbers of patients are not given in the English summary.] All the cases of malignant tertian were given 0·06 gram daily, and they all relapsed. (Compare MANIFOLD, above, p. 1007.)

A drug named Saiko, which has long been used by the Chinese in malaria and other fevers, was also tested. Botanically it includes three different herbs, *Potentilla chinensis* (Rosaceae), and two umbelliferous plants. It had no effect upon the malaria parasites, and the authors conclude that it is useless.

W. F.

MORISHITA (Kaoru) & NAMIKAWA (Hiroshi). **Studies in the Treatment of Malaria. IV. Therapeutic Effect of Osvarsan upon Various Forms of Malaria. V. Influence of Jozan-Decoot upon Malarial Infection.**—*Taiwan Igakkai Zasshi* (*Jl. Med. Assoc. Formosa*). 1931. July. Vol. 30. No. 7 (316). [In Japanese. English summaries pp. 55 & 56.] [Govt. Research Inst., Formosa.]

Osvarsan is an oral antisyphilitic drug, made in Japan, with the same constitution as stovarsol, and the chemical definition 4-oxy-3-acetylamino-phenylarsinic acid. The authors found that it caused disappearance of both the parasites and fever of benign tertian malaria, but it did not prevent subsequent relapses. It had no destructive action upon subtertian or quartan parasites. Daily doses of 0·75 to 0·8 grams were given, either for a week continuously or for two periods of 3 days with an interval of 4 days. In one case, where 0·8 gram was given every day, severe intoxication occurred with fever, exanthem, and cerebral symptoms.

Under the name Jozan a number of plants is used in the Far East as a remedy for malaria. The two plants most commonly used are *Orixa japonica* and *Hydrangea macrocephala*. These were administered to cases of all the three forms of malaria, without any apparent effect.

W. F.

DE NORONHA (Remo). Acção da plasmoquina nas esplenomegalias palustres. (Elementos clinicos.) [**Effect of Plasmoquine on Malarial Splenomegaly.**]—*Arquivos da Escola Méd.-Cirurg. de Nova Goa.* Ser. A. 1931. No. 6. pp. 959–976.

Details are given of 13 patients with enlarged spleens from malaria infection, 6 treated with plasmoquine 3 tablets daily, 6 with plasmoquine compound, 12 tablets daily in 3 doses of 4 each ; in one case the form of the drug is not stated.

The final note as regards the spleen was made about 6 months after starting treatment. Of the 6 who were given plasmoquine only, in four the spleen was completely reduced. Of the 6 who had the compound, in one the spleen was reduced to normal, in one there was considerable reduction, in three it was softened to the feel, but not reduced in size to any extent.

[In judging the results other factors would have to be taken into account, but, regarding the splenomegaly alone, the above would seem to show that simple plasmoquine had the greater effect.]

H. H. S.

MANSON-BAHR (Philip). **The Chemoprophylaxis of Malaria.** [Correspondence.]—*Lancet.* 1931. Aug. 22. pp. 425–426. [6 refs.]

Dr. Manson-Bahr draws attention to the debt of gratitude which workers in tropical medicine owe to Colonel JAMES and his co-workers

for their demonstration of the value of plasmoquine* as a prophylactic. Dr. Manson-Bahr, for the last three years, has advocated the use of plasmoquine compound as a prophylactic to patients returning to malarious places in the tropics. It is his practice to prescribe one tablet of plasmoquine compound (plasmoquine 0·01 gram, quinine sulphate 0·125 gram), last thing at night during the malaria season, but never on an empty stomach. The results have been most satisfactory.

W. F.

REHBEIN (Max). Heilung eines Falles von Malaria tertiana mit Chininidiosynkrasie durch Plasmochin. [**Cure of a Case of Benign Tertian Malaria with Quinine Idiosyncrasy by Plasmochin.**]—*Arch. f. Schiffs- u. Trop. Hyg.* 1931. July. Vol. 35. No. 7. pp. 435–436. With 1 fig.

Patient had received plasmochin compound for the treatment of malaria and developed haemorrhages under the skin and bleeding from various mucous membranes; when he was placed on pure plasmochin without quinine the symptoms did not develop and he got rid of the malaria. The author considers this a case of idiosyncrasy to quinine.

E. D. W. Greig.

RUSSELL (Paul F.). **Plasmochin Simplex, a Prophylactic Drug in Avian Malaria. Preliminary Report.**—*Amer. Jl. Trop. Med.* 1931. July. Vol. 11. No. 4. pp. 279–284. With 3 text figs. [3 refs.] [Bureau of Science, Manila.]

Plasmochin simplex was administered daily to canaries for a period of seven days by means of intramuscular injections of 0·00016 to 0·0002 grams. The birds were inoculated on the third day, and some of them on later days, with blood containing the parasites of avian malaria. None of them developed malaria except a pair inoculated on the seventh day, a few hours after having received their last dose of plasmochin. On the contrary, canaries similarly treated with 0·005 gram doses of quinine all developed malaria.

W. F.

DE LA CAMARA (Pedro) & MORALEDA (Carmen). Observations de trente et un cas de paludisme traités par le quiniostovarsol. [**Thirty-One Cases of Malaria treated with Quiniostovarsol.**]—*Bull. Soc. Path. Exot.* 1931. Apr. 15. Vol. 24. No. 4. pp. 335–356. With 3 graphs. [13 refs.] English summary.

——. Nota sobre la eficacia del quiniostovarsol en la prevención de las recidivas. [**The Efficiency of Quiniostovarsol in preventing Malarial Relapses.**]—*Medicina Países Cálidos.* Madrid. 1931. Sept. Vol. 4. No. 5. pp. 426–429. [6 refs.] English summary (5 lines). [Antimalaria Inst., Navalmoral de la Mata, Cáceres, Spain.]

The author, with Moraleda, reported [*ante*, p. 601] on 31 cases of malaria treated with quiniostovarsol, dealing with its effects in

* An editorial footnote to Dr. Manson Bahr's letter is as follows: "There has been some confusion about the name and composition of this drug. We are assured by Bayer Products, Ltd., the London agent of its makers, that Plasmoquine, as now issued, is identical with the preparation in the first instance issued under the name of Beprochin. There has been no alteration in its composition. Plasmochin is simply the continental spelling of Plasmoquine. The makers state that Plasmoquine is N-diethylamino-isopentyl-8-amino-6-methoxy-quinoline, obtained synthetically. It is not derived from quinine.—ED. L."

expelling parasites from the circulation, in reducing temperature and splenomegaly, etc. In this paper are described the conditions of the patients 8 months after the treatment ceased. Of 15 with benign tertian infection (one patient is excluded, having shown intolerance of the drug) 9 had relapsed. These relapses could not be attributed to inadequate dosage or courses, for only two of the nine had failed to complete the third course of ten days, whereas three of the six who did not relapse received only two courses. Among 11 patients with subtertian malaria none had relapsed, although one had previously relapsed after plasmoquine co. and two after quinine. The conclusion is drawn that quiniostovarsol is very rapidly effective, as regards its immediate action, on *P. vivax*, but that relapses occur in the majority after a varying interval; on *P. falciparum* the action is slower but more complete seeing that none of the patients so infected relapsed.

H. H. S.

i. MONIER (H. M.). Essai d'un dérivé de la quinoléine (664 Fourneau) dans la malaria expérimentale. [**A Trial of Fourneau 664, a Quinolin Derivative, in Experimental Malaria.**]—*Bull. Soc. Path. Exot.* 1931. Feb. 11. Vol. 24. No. 2. pp. 93–97.

ii. ——. Essais thérapeutiques du 710 Fourneau dans quelques cas de paludisme. [**Fourneau 710 in the Treatment of Malaria.**]—*Ibid.* pp. 97–101. With 2 charts in text.

iii. ——. Action de la plasmoquine simple dans un cas de paludisme à *P. praecox*. [**Plasmoquine in a Case of Subtertian.**]—*Ibid.* pp. 101–104. [1 ref.]

i. Fourneau 664 is allied to plasmoquine. Eight general paralytics who had been therapeutically infected with benign tertian, were treated with intramuscular injections of the drug. The author concludes that it has an action on the benign parasite, that 0·04 gram a day is insufficient, and that 0·1 gram is dangerous. [The drug is evidently unsatisfactory].

ii. Fourneau 710, a yellow powder readily soluble in water, proved very active against *P. vivax*; in one case, a single dose of 0·04 gram was enough to clear the blood of parasites. It had no action in subtertian.

iii. A patient, intolerant of quinine, was successfully treated with plasmoquine.

W. F.

MONIER (H. M.). Association du 710 Fourneau avec la quinine et le stovarsol. [**Association of Fourneau 710 with Quinine and Stovarsol.**]—*Bull. Soc. Path. Exot.* 1931. May 13. Vol. 24. No. 5. pp. 378–382. [1 ref.]

Good results were obtained in 4 cases of subtertian and 2 of benign tertian. The maximum dose of 710 Fourneau is 0·02 gm., and the optimum amount to be given in one day is between 0·06 and 0·08 gm. After a week's treatment there should be a week's rest. Larger doses may cause violent headache, shivers and sweats.

W. F.

SERGENT (Edm.), SERGENT (Et.), CATANEI (A.), TRENSZ (F.) & SERGENT (A.). Etude de l'action du "710" Fourneau sur le paludisme des oiseaux à *Plasmodium relictum* (note préliminaire). [**The Action of "710 Fourneau" upon Bird Malaria.**]—*Ann. Inst. Pasteur.* 1931. July. Vol. 47. No. 1. pp. 57–62. [4 refs.] [Pasteur Inst. of Algeria, Algiers.]

Canaries to the number of 533 were employed in these tests. A single dose of 0·00025 gram of 710 may kill a canary, but if it is given in divided doses during the day it will not. Similarly a single dose of 0·002 of quinine may be fatal, but it is harmless in divided doses. The curative dose of 710 is only one-tenth of the fatal dose, but the curative dose of quinine is almost as high as its fatal dose. In other respects, both in the cure and the prevention of the inoculated malaria of canaries, the results of treatment with 710 are practically the same as those obtained with quinine.

W. F.

WARSTADT (A.) & COLLIER (W. A.). Ueber die klinischen Ergebnisse der Behandlung Malariakranker mit den Präparaten "R 118" und "R 123". [**Clinical Results of Treatment of Cases of Malaria with "R 118" and "R 123."**]—*Ztschr. f. Hyg. u. Infektionskr.* 1931. May 22. Vol. 112. No. 3. pp. 534–543. [Robert Koch Inst. for Infectious Diseases, Berlin.]

The two drugs are slight modifications of a chinolin preparation produced by Dr. A. ROTHMANN and Dr. K. FRICKER of the Chemical Institute of the University of Berlin. The authors conclude that the two preparations are harmless in relatively high doses. They have never observed cyanosis or other complication. The cases treated were chiefly general paralytics inoculated with *P. vivax* and *P. malariae* and some cases of natural infection. They find that the therapeutic action in malaria is relatively slow, but in inoculation malaria, both tertian and quartan, a favourable result was produced. Also in natural infections with these parasites, not only the schizonts but also the gametocytes were destroyed. Further investigations, carried out over a long period and under suitable conditions, will be required before they can express an opinion as to complete sterilization of the organism. Also the most suitable dose will have to be determined. The drugs were administered intramuscularly, subcutaneously and orally.

E. D. W. Greig.

FERRADAS (Manuel G.). La adrenalina como coadyuvante en el tratamiento del paludismo. [**Adrenalin as an Auxiliary in the Treatment of Malaria.**]—*Medicina Países Cálidos.* Madrid. 1931. May. Vol. 4. No. 3. pp. 243–244.

When engaged on an Anti-malarial Campaign in 1928 the author gave to suspected subjects (those with enlarged spleen, anaemia and debility) a dose of adrenalin in order to detect as many infected as possible and with a view to treating them before the next hatching out

of mosquitoes. Though the results as regards " activation of parasites" were disappointing the patients showed notable improvement in their general condition. Seeing that certain of the symptoms accompanying plasmodium infection—weakness, prostration, low pulse tension, vomiting, anaemia, etc.—may be due to adrenal deficiency, he started giving 20 drops of 1 in 1,000 adrenalin daily 4 hours after the quinine for 10 consecutive days, and, after a 10 days' interval, another course of 8 days. He found that the quinine was more easily tolerated than when given alone, and that headache and vomiting rapidly disappeared and the general state was much improved. The number so treated has been up to the present few, but the author states that of 15 primary cases treated in 1928 only 2 have relapsed up to 1930, whereas 65 per cent. relapsed [total not given] of those treated by quinine alone. Of 22 chronic patients (3 years or more) with anaemia and splenomegaly, only 6 relapsed and in all the cachectic signs cleared up. The adrenalin, he maintains, " enhances the effect of quinine, raises the patients' tolerance and has [general] beneficial effects."

H. H. S.

DILIBERTO (Ugo). Tentativi di terapia medica delle splenomegalie malariche croniche. [**Medicinal Treatment of Chronic Malarial Splenomegaly.**]—*Riv. Sanitaria Siciliana.* 1931. May 15. Vol. 19. No. 10. pp. 711–714, 717. [4 refs.] English summary (5 lines). [General Med. Clinic, Univ., Palermo.]

Details of six patients are recorded. In all the spleen was enlarged to the level of the umbilicus, and each patient had suffered from malaria for several, 4 to 16, years. The author's method was to give adrenalin intravenously, daily or every other day according to the degree of reaction, in doses starting with 0·01 mgm. and increasing gradually to 0·1 mgm. or, rarely, 0·2 mgm. The usual duration of treatment was about 2 months, and some 40 injections were given in each case totalling 3·5 mgm. of adrenalin. In two of the patients the spleens returned to the normal size ; in three others there was marked reduction to one half or one third ; one failed altogether to respond and the treatment was abandoned after 40 injections had been given (3·5 mgm. in all) in 2 months. Susceptible patients exhibited pallor, headache, tremor, muscular irritability and mental excitability. In all but one there was marked improvement in the anaemia. The red cells increased in one patient from 625,000 to 4,000,000 and the Hb from 40 to 80 per cent., in another from 900,000 to 2,000,000 and Hb 50 to 70 per cent. ; those who showed most reduction of spleen also showed greatest improvement in the blood condition.

H. H. S.

SURBEK (K. E.). Zur Frage der Splenektomie bei gewissen Fällen von chronischer Malaria. Dauerresultate. [**Splenectomy in Certain Cases of Chronic Malaria. Lasting Results.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1931. Sept. Vol. 35. No. 9. pp. 537–540. [1 ref.] [Central Pagar Alam Hosp., Sumatra.]

The author performed splenectomy in 10 cases of enlarged spleen in chronic malaria. Although parasites were found in only one blood before operation it was assumed that there could be no question of the

splenomegaly in Java and Sumatra being due to any other disease. The operation was done under local anaesthesia. The weight of the spleens removed varied from 250 to 1,250 gm., the ages of the patients from 12 to 29 years ; 9 were males and one a female. The operation was undertaken after several weeks' observation, during which the usual combination of quinine and arsenic had proved unsatisfactory. Plasmochin was not available. Apparently the patients failed to recover on account of the large spleen ; unfortunately the author was unable to confirm this view by haematological investigations. The permanent after-results were satisfactory. Medical treatment now produced much better results. The anaemic condition improved and the patients lost the pale icteric look. In two cases a slight renal disturbance cleared up.

E. D. W. Greig.

UMANSKY (James). **Treatment of Malarial Coma.**—*Lancet.* 1931. Aug. 15. pp. 349–350. [1 ref.]

The author treated 14 cases of malarial coma at Doshambe, in the subtropical country of Tagykistan. The first 4 died, but the other 10 treated by injections of 3 cc. of a 40 per cent. solution of urotropine, sterilized by boiling, all recovered. The cerebral symptoms passed off in a few hours ; the pyrexia and parasites remained, and were dealt with by the usual therapy. He suggests that the formaldehyde derived from the urotropine hinders the adherence of the damaged erythrocytes to the walls of the blood vessels and restores the circulation in the blocked capillaries.

W. F.

BARRAUD (P. J.). **Notes on Some Entomological Technique for the Malariologist.**—*Records of the Malaria Survey of India.* 1931. Mar. Vol. 2. No. 1. pp. 157–160..

1. The transmission of Anopheles larvae through the post.

" The following method for sending specimens of larvae through the post has been tested during the past year and the results have proved very satisfactory. A specimen tube 2½ inches × 1 inch, or 3 inches × 1 inch, is prepared by pushing a wad of cotton-wool, soaked in formalin, down to the bottom, and this is held firmly in place by a disc of cork. The disc should be about ¼ inch thick as, if it be too thin, it will warp and shake loose. The disc may conveniently be cut from the lower end of the cork supplied with the tube, but care must be taken to see that it fits the tube very tightly. The wad of cotton-wool should be about ½ inch thick when firmly pressed down. Sufficient formalin should be used so that when the cork disc is forced down on to the wool it will become saturated with the liquid, any excess being afterwards poured off.

" Larvae are transferred to the tube, from water, either with a spoon or with a wide-mouth pipette ; 30 or 40 larvae may be placed in one tube. *All the water* is then poured off, or drawn off with a pipette, leaving the larvae stranded on the cork disc or on the sides of the tube. The open end of the tube is then firmly corked, the tube done up in cotton-wool, or soft paper, and packed in a wooden box. The larvae are very soon killed by the formalin vapour and remain attached to the cork disc, or

the sides of the tube, quite firmly, and there is very little risk of their being shaken about."

2. Rearing Anopheles from the egg.

" . . . Gravid female *Anopheles* are selected and confined in lamp globes. For single specimens 'Boy Globes,' 4 inches × 2¼ inches, obtainable in Calcutta, are very suitable; for numbers of specimens ordinary hurricane lamp globes are used. The upper end of the globe is covered with mosquito netting upon which are placed some raisins and a strip of lint, the latter being kept continuously damp. A receptacle containing water is placed inside the lower end of the globe. Some grass stems may be pushed into the globe beforehand to afford resting places for the mosquitoes. The globes should be kept in a dark place free from ants. Should the temperature be very low, the globes may be kept in an incubator at about 20° to 22° C. . . . The gravid mosquito is transferred to a test tube and then allowed to fall from the tube on to the surface of water. Frequently it will commence to lay within a few minutes. If the mosquito be very active, it may be partially stunned by rapping the tube smartly against the hand and the mosquito then allowed to fall on to the water.

" Soon after the eggs have been laid, they are transferred with a camel-hair brush to water in a bowl, basin or dish. The best results have been obtained with glass petri dishes 8 to 10 inches in diameter."

The eggs are floated within a ring of cork, coated with hard paraffin, which prevents the eggs from becoming stranded on the side of the dish.

" Some *Spirogyra* and a plant of grass, with some soil attached to the roots, are added, and, as soon as the larvae hatch, some chopped flies are scattered on the surface of the water. The *Spirogyra* should be kept in a basin for some time previously and exposed to sunshine, search being made from time to time for any eggs, larvae, or other living creatures and these removed. *From now onwards the dishes containing the larvae are kept in the sun, during the early morning hours, or for several hours during the day.* This is the most important part of the technique. The sun causes bubbles of oxygen to form in the *Spirogyra* which keep the water aerated and prevent a scum forming on the surface. The grass plant affords shade and shelter to those larvae which prefer this condition to direct sunlight.

" If convenient, the dishes are examined about midday, when some cool water and more food material are added if necessary. About 4 or 5 p.m. the dishes are put away for the night and are covered with another petri dish, or sheet of glass to prevent other mosquitoes from laying eggs upon the water. It should be mentioned that on no account should the dishes be covered when exposed to sunshine.

" The time occupied in rearing larvae by this method is usually not more than 15 to 30 minutes daily, according to the number of dishes to be handled, and the work can be done by an assistant with occasional supervision."

W. F.

ANAZAWA (K.). **Experimental Studies on the Infectability of Anopheline Mosquitoes of Formosa.**—*Taiwan Igakkai Zasshi* (*Jl. Med. Assoc. Formosa*). 1931. June. Vol. 30. No. 6 (315). pp. 609–632. [84 refs.] [In Japanese. English summary pp. 42–45.]

The mosquitoes employed were bred from larvae in the laboratory. They were fed only once on a carrier, and were then kept in cages at an average temperature between 20° and 29° C. The results were as follows :—

1. Result of experimental infection with tertian fever :

Species of Anopheles.	Number of mosquitoes dissected after feeding.	Number of mosquitoes with developed oöcysts.	Number of mosquitoes with developed sporozoites (infected salivary glands).	Total infected.	Percentage of infection.
A. sinensis ...	42	24	8 (2)	25	59·5
A. minimus	23	15	5 (1)	15	65·2
A. maculatus	11	6	4 (1)	7	63·6
A. tessellatus	2	2	—	2	100
A. hatorii ...	13	8	4 (1)	8	61·5
A. fuliginosus	16	9	3	9	56·3
A. splendidus	7	2	3 (1)	4	57·1
A. pleccau ...	5	4	3	4	80

2. Result of experimental infection with quartan fever :

Species of Anopheles.	Number of mosquitoes dissected after feeding.	Number of mosquitoes with developed oöcysts.	Number of mosquitoes with developed sporozoites (infected salivary glands).	Total infected.	Percentage of infection.
A. sinensis ...	202	34	—	34	16·8
A. minimus ...	93	11	—	11	11·8
A. maculatus	28	9	—	9	32·1
A. tessellatus	12	6	3 (1)	6	50
A. hatorii ...	26	4	—	4	15·4
A. fuliginosus	64	12	3	13	20·3
A. splendidus	14	5	—	5	35·7
A. pleccau ...	13	6	1	6	46·2

3. Result of experimental infection with subtertian fever :

Species of Anopheles.	Number of mosquitoes dissected after feeding.	Number of mosquitoes with developed oöcysts.	Number of mosquitoes with developed sporozoites (infected salivary glands).	Total infected.	Percentage of infection.
A. sinensis ...	29	0	0	0	0
A. minimus	28	18	3 (2)	18	64·3
A. maculatus	23	11	3 (1)	11	47·8
A. tessellatus	26	23	10 (3)	23	88·5
A. hatorii ...	31	21	—	21	67·7
A. fuliginosus	9	7	3 (1)	8	88·9
A. splendidus	15	1	3 (3)	4	26·7
A. pleccau ...	5	5	1 (1)	5	100

W. F.

SWELLENGREBEL (N. H.) & DE BUCK (A.). **Correlation between Intestinal and Salivary Infection in** *Anopheles maculipennis.*—Reprinted from *Proc. Roy. Acad. Sci. Amsterdam.* 1931. Vol. 34. No. 1. pp. 183–185. With 1 text fig. [Royal Colonial Inst., Amsterdam.]

In 23 batches of *A. maculipennis*, infected with benign tertian malaria, containing an average of 59 in each batch, the mean oöcyst-rate was 45 per cent. and the mean sporozoite rate 47 per cent. "This unambiguous result should remove all doubt as to the accuracy of the oöcyst-rate as a measure to test the suitability of *A. maculipennis* to transmit malaria." The authors maintain that the sporozoite rate is less accurate than the oöcyst rate. They conclude that, in *A. maculipennis*, intestinal infection provides a more accurate estimate of the number of sporozoite carriers than salivary infection, and this probably applies to other species as well.

W. F.

AVERBOUCH (Ida). Expériences sur la transmission à l'homme [de] la fièvre quarte par les piqûres d'*Anopheles maculipennis* var. *sacharovi*. [**Experimental Transmission of** *P. malariae* **to Man by** *A. maculipennis* **var.** *sacharovi*.]—*Rev. Microbiol., Epidémiol. et Parasit.* 1930. Vol. 9. No. 3. pp. 379–381. [4 refs.] [In Russian. French summary.]

Working in Tashkent the author succeeded in infecting *Anopheles maculipennis* var. *sacharovi* from a case of quartan malaria and in transmitting the infection to another human being. The experiments were conducted in the winter of 1928–1929. The mosquitoes were collected in their hibernating quarters and kept in glasses covered with muslin. The patient on which the mosquitoes were fed had the first attack in October, 1928; when first examined ten days later, *P. malariae* was found in the blood. He was given injections of 50 per cent. solution "chinopyrini" (?) in doses of 2 cc. until the symptoms disappeared. About a month later there was a relapse and treatment was resumed, but failed to stop the attacks. In the middle of December, when the patient still showed a fair number of parasites, the author allowed 96 *A. maculipennis* var. *sacharovi* to feed on herself. From the 14th day onwards zygotes were found in some of the mosquitoes, but no sporozoites. On the 26th day only three mosquitoes remained alive, and these were allowed to feed on the author. A month later she had the first attack. Parasites (*P. malariae*) were recovered during the third attack. About a fortnight later gametes appeared in the blood. During the whole of this period the patient remained without treatment. The author notes that at the time of infection she was undergoing a course of antirabic inoculations and believes that these may have played a provocative rôle in the development of the malarial symptoms.

C. A. Hoare.

STRICKLAND (C.) & ROY (D. N.). **The Value of the "Sergents' Method" for detecting Malarial Infection in Mosquitoes.**—*Indian Med. Gaz.* 1931. July. Vol. 66. No. 7. pp. 388–390. With 2 text figs. [5 refs.]

The authors have followed the technique recommended to them by Dr. Edmond SERGENT, with slight modifications. After pulling off

the legs and wings, the mosquito is placed on a slide and decapitated with a sharp-edged needle. The thorax is then pressed gently with a blunt needle so that some body-fluid exudes from the cut end into a minute drop of salt-solution. The S-shaped or sickle-shaped sporozoites, which are about 14μ long, can be found with the 1/6 objective. The authors always found sporozoites by this method when they were present in the salivary glands, and if there were none in the body-fluid, there were none in the salivary gland. This method is far quicker and far more simple than dissecting out the gland.

W. F.

CARLEY (Paul S.). **Results of the Dissection of 1,017 Wild-Caught Anophelines in Jamaica.**—*Amer. Jl. Trop. Med.* 1931. July. Vol. 11. No. 4. pp. 293–296. With 1 text fig. [1 ref.]

As the result of the dissection of 1,017 wild Jamaican anopheles, two specimens of *A. albimanus* were found infected, one in the stomach and one in the salivary glands. One specimen of *A. grabhami* contained a single oöcyst.

W. F.

MESNARD (J.) & MORIN (H.). Réceptivité naturelle de *Anopheles* (*Myzomyia*) *aconitus* à l'infection par l'hématozoaire du paludisme en Cochinchine. [**Natural Infection of** *A. aconitus* **in Cochin China.**]—*Bull. Soc. Path. Exot.* 1931. July 8. Vol. 24. No. 7. pp. 554–556.

The authors have found that in Cochin China *A. aconitus* is infected to the same degree as *A. minimus* ; the latter anopheline is however far more numerous.

W. F.

KNOWLES (R.). **The Laboratory Diagnosis of Malaria.**—*Indian Med. Gaz.* 1931. May. Vol. 66. No. 5. pp. 271–278. With 3 text figs. [12 refs.]

This is an address to the Calcutta Branch of the B.M.A. Colonel Knowles always examines both thick and thin films in every case. In examining thin films he uses a 1/7th inch fluorite immersion lens, switching over to the 1/12th inch when necessary. The following method is used for thick films.

Flood the slide with this mixture : Glacial Acetic 2·5 per cent. in distilled water, 4 parts ; Crystal Tartaric Acid 2 per cent. in distilled water, 1 part.

This dehaemoglobinizes the film which becomes grey white.

When dehaemoglobinization is complete, pour off the fluid and flood with methyl alcohol, for 5 minutes.

Drain off the methyl alcohol and wash very thoroughly with neutral or slightly alkaline distilled water.

Stain with Giemsa.

The distilled water must be neutral or faintly alkaline. To ensure this, put 5 cc. in a test tube and add 5 drops of a 0·04 per cent. watery solution of bromo-cresol purple. If the water is acid it will turn pale yellow. If it does so, add a 1 in 1,000 solution of sodium carbonate, drop by drop, until the water just turns violet, which indicates that it has become faintly alkaline. Methyl alcohol rapidly loses its fixative power in the tropics. It should be put up in sealed 5 cc. phials, and should be specified as "*purissimum*, free from acetone."

Bass's blood culture method has been employed for the last two years, in Calcutta, as a routine method of diagnosis, and it detects infections in which the parasites are too scanty to be found in films. The following technique is employed.

Five cc. of blood, drawn off with a sterile, perfectly dry syringe, are put into an Ehrlenmeyer flask containing 20–30 glass beads, and are defibrinated by gentle rotation of the flask on the laboratory bench. Care must be taken not to form bubbles. A 50 per cent. solution of Merck's dextrose *purissimum* is sterilized by steaming for half an hour on each of three successive days. One drop of this solution is put into each of a number of small test tubes measuring 12½ by 1¼ cm. The defibrinated blood is pipetted into these tubes, so as to form a column about 2½ cm. deep in each. The upper parts of the tubes are warmed and, while they are still warm, rubber teats are fitted over their mouths to insure partial anaerobiosis. They are incubated at 37° C. and examined after 12, 24, and 48 hours (see this *Bulletin*, 1930, Vol. 27, p. 198, and Vol. 10, p. 169).

W. F.

SIEBURGH (G.). De kleuring van malaria parasieten volgens Leishman-Shute. [**The Malaria Parasite Staining Method of Leishman-Shute.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1931. Aug. 15. Vol. 71. No. 10. pp. 904–909. [26 refs.] [Med. Lab., Batavia.]

Extending the staining period from 30 to 45 minutes Sieburgh otherwise exactly followed SHUTE's technic (see this *Bulletin*, Vol. 27, p. 663). He could show neither the quartan stippling in younger stages of the quartan parasite, nor Schüffner's dots in the youngest stages of tertian parasites. Maurer's spots in subtertian were regularly found. This failure, which possibly may be ascribed to influences of the tropical climate on the staining procedure, does away with the practical advantage of SHUTE's method, i.e., the easier identification of the younger stages of the parasites.

W. J. Bais.

GARNHAM (P. C. C.). **The Staining of Malaria Parasites.**—*Kenya & East African Med. Jl.* 1931. May. Vol. 8. No. 2. pp. 56–57.

It is difficult to distinguish subtertian from quartan rings. The difficulty can be overcome by using a stain which demonstrates Maurer's spots in the former. Acidity of the slides, which interferes with correct staining, can be avoided by treating them with a saturated solution of sodium silicate, washing and drying. To demonstrate Maurer's spots, the following stain is recommended :—

Eosin methylene blue	0·32 gram.
Eosin methylene violet	0·16 gram.
Methyl alcohol (acetone free)	100·00 cc.

Leave the stain in a bottle for 3 days after mixing and then filter. To use, fix a thin film with 2 or 3 drops of the stain for not longer than 5 seconds ; add 10 times the quantity of distilled water ; leave for ¾ hour ; flush with distilled water ; dry and examine. Leucocytes and crescents are grossly overstained and the slide is covered with a light deposit, but infected cells are clearly shown.

W. F.

BRINKMANN (Ernst). Schneller Nachweis der Malariaplasmodien. [**Method for Rapid Detection of Malarial Plasmodia.**]—*Med. Klin.* 1931. May 1. Vol. 27. p. 661.

This procedure is practically the same as the ordinary method of vital staining for reticulocytes. A 2 per cent. alcoholic solution of brilliant cresol blue is allowed to dry on a slide forming a patch about the size of a shilling. A small drop of blood is applied to the middle of the patch and

covered with a cover glass; the blood should not spread out quite to the edge of the cover. The author recommends practice with typical cases of malaria to familiarize the appearances of the parasite with the new method. He claims that it is extremely simple and rapid, allowing the examination of a comparatively thick drop of blood. Positive results are more frequently and earlier obtained than by other methods. The disadvantage is that permanent preparations cannot be made.

E. D. W. Greig.

ROCCHI (Filippo). Intradermoreazioni e anticorpi verso il pigmento malarico. (**Intradermic Reactions and Elaboration of Antibodies against Malarial Black Pigment.**)—*Riv. di Malariologia.* 1931. Mar.-Apr. Vol. 10. No. 2. pp. 161–182. [13 refs.] English summary p. 266. [Inst. of Path. Anat., Univ., Rome.]

It would require a very full abstract, almost a translation, to deal adequately with the points brought forward in this article and those who have been studying and working at serological reactions in malaria should consult the original. The author enumerates the various attempts which have been made to find a satisfactory antigen for complement fixation tests in malaria and discusses in greater detail HENRY's ferro- and melanoflocculation. He then states that his aim was to find out if there was not in a malaria subject some antibody to melanin, using malaria pigment itself as antigen; his test differs therefore from Henry's in the employment of malaria pigment instead of choroidal pigment or a proteinate of Fe. It differs also in not being an extracorporeal but an intradermal reaction. From it one infers that malarial pigment is not an inert substance, but acts as an endoantigen.

The author describes in detail his technique for obtaining the pigment and for the test suspends it in a fluid which he found after many trials gave no skin reaction by itself, namely, saturated solution of lithium carbonate 1·3 parts, physiological saline 10 parts. The amount injected is 0·5 cc. He expected to find a positive result in malarial subjects and negative in non-malarious controls, and to his surprise the reverse occurred. The size of the wheal may be 55 x 30 mm., but the average was 30 x 25 mm., beginning to show itself not at once but after 4–5 hours, lasting for 15 or, occasionally, 48 hours.

The test was carried out on 105 malarious subjects, subdivided into groups: 1. Primary cases of benign tertian (23); 2. Relapsed cases of benign tertian (30); 3. Primary malignant tertian (24); 4. Relapsed m.t. (17); 5. Quartan, 2 primary and one relapsed; 6. Chronic malaria (8); also of three general paralytics inoculated with b.t. and of 15 persons healthy or suffering from non-malarial disease but cured many years before of malaria. For controls he tested 109 persons, either healthy or suffering from disease other than malaria and giving no history of any malaria in the past.

Here only an epitome of the results can be given which are more fully described in the article. Of the 105 with malaria actually present 80 were negative (76 per cent.); of the 15 with old cured malaria 14 were positive; of the 109 controls 90 were positive (82·5 per cent.); lastly, the general paralytics gave a positive before being inoculated with *P. vivax*, negative after and during the time of febrile attacks, returning again to positive 40–60 days later. The test would seem, therefore, to bear an analogy with the Schick test for diphtheria. The pigment, in short, provokes a local reaction in a great majority of non-malarious subjects and those who have been cured of the disease,

but leads to no response in those actually suffering from it. As a final test the author took blood from a patient with numerous parasites and from a healthy individual, filtered them separately (to remove all parasites from the former) and diluted the sera equally; he then injected intradermically two non-malarious subjects with 2 cc. [sic] of each in corresponding situations. The local swellings produced subsided in 2–3 days and on the fifth day he injected the pigment solution into one zone and haemoglobin solution into the other. [This was done in all the tests, but has been omitted from the above description in order not to complicate matters; the reaction to Hb was a weak positive in practically every case.] By way of control these two solutions were injected into another individual whose skin had not been previously treated. The latter gave + + +; the normal serum areas on the first two gave a + + to the injected pigment, the "malaria serum" areas a negative, thus demonstrating the passive transmission of local skin immunity from a malarious to a non-malarious subject. Five days later this local reaction became lost.

H. H. S.

BROTZU (G.). Valeur des réactions floculantes dans la diagnose de la malaria. [**The Value of Flocculation Reactions in the Diagnosis of Malaria.**]—*Boll. Sezione Ital., Soc. Internaz. di Microbiologia.* Milan. 1931. July-Aug. Vol. 3. No. 7–8. pp. 429–433.

——. Valore delle reazioni flocculanti nella diagnosi di malaria.—*Atti III. Congresso Naz. di Microbiol., Milano, 1931.* pp. 77–81.

There are two malaria pigments, namely melanin and an ochreous pigment which is rich in iron. HENRY (*ante*, p. 139) could not obtain these pigments from the human body in sufficient quantity to serve as antigens in his reactions, and consequently he substituted choroidal pigment for the first, and methylarsinate and albuminate of iron for the second. The author employs yet another antigen, namely sepia, diluted until it is brown and limpid. In 32 cases where the choroidal pigment gave a positive reaction, the sepia was positive in 28. He considers the choroidal antigen too sensitive. His conclusions are as follows:

The reaction appears quickly in the first attack of malaria. It is sometimes negative during the fever. It may be positive in persons not having attacks, whether there are parasites in their blood or not. The reaction remains positive for a certain time during the cure and, in some cases, even after. The reactions with choroid pigment and with sepia are more sensitive than the reactions with iron salts. In thirteen controls, suffering from diseases other than malaria, there were no positive reactions.

W. F.

LAVERGNE (J.) & MONIER (H.). Utilisation de la sérofloculation de Henry dans le diagnostic et le traitement du paludisme. [**Henry's Reaction in the Diagnosis and Treatment of Malaria.**]—*Bull. Soc. Path. Exot.* 1931. July 8. Vol. 24. No. 7. pp. 539–544. [1 ref.]

In 16 cases with fever and parasites, the reaction was positive in only 10. In 2 cases where malarial infection was unlikely, there were strong positive reactions. In 36 cases of probable latent malaria, there

were 22 positive reactions. In 49 cases with no symptoms or history of malaria, the reaction was negative in all. The author concludes that the reaction is clearly specific, but that it would be more accurate if readings were taken with a photometer rather than with the naked eye. Some patients give a much stronger reaction than others, independently of their condition and of the kind of parasite with which they are infected. Many of the cases were examined for leprosy by means of the sedimentation test for leprosy with formolized sheep cells, and, in four cases where the Henry reaction was very strongly positive, there was an exceptional quantity of agglutinins for sheep's corpuscles which disappeared when the Henry reaction became negative.

W. F.

HENRY (F. X.). Malaria-floculation; les antigènes; l'observation macroscopique des résultats. [**Malaria Flocculation: Antigens: Reading of Results.**]—*C.R. Soc. Biol.* 1931. Sept. 18. Vol. 107. No. 26. pp. 1520–1522. [1 ref.]

The author deals with certain technical points and modifications in connexion with his reactions. When métharfer gives a positive reaction the melanin reaction is positive too; in consequence, he has abandoned the former, and uses only one iron salt—an albuminate of iron No. 113, specially prepared by Merck. For routine diagnosis, he considers it sufficient to use melanin as the only antigen. The tubes are kept in the incubator for 2½ hours; the albuminate tubes are read at once, and the melanin tubes after ½ an hour at room temperature. The precipitate in the albuminate tubes is finer and more granular than that in the melanin tubes. He insists upon the importance of reversing the tubes gently, twice, before reading the results, in order to distinguish flocculation from simple deposition. The results can be read with the naked eye, if the tube is held at an angle before a lamp placed slightly above the level of the observer's eyes. In order to bring the melanin antigen to suitable strength, it should be diluted until its turbidity matches that of 0·20 gram of albumin per litre. Acid-distilled water must be avoided. The water should be doubly distilled in the presence of glass beads.

W. F.

ROW (R.). **Precipitin Reaction in Malarial Sera.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1931. Apr. 25. Vol. 24. No. 6. pp. 623-627. With 1 plate. [8 refs.] [Grant Med. College, Bombay.]

Cultures of malaria parasites are used as antigen. First the supernatant serum is removed and the deposit, consisting of merozoites and corpuscles, is washed three times with salt solution. It is then laked in distilled water and centrifuged at high speed. The resulting woolly deposit is dried at 37° C. and rubbed up with 20 times its volume of normal saline: this is the antigen. The test is carried out in tubes of 0·5 cm. bore. A column of the test serum is put into a tube, and an equal quantity of antigen is carefully floated on to the top of it, without mixing. In positive cases, a ring of opalescence appears, after 3 or 4 hours incubation at 37° C., which deepens after 18 hours at room temperature. The reaction is chiefly a group reaction; the phenomenon is common to sera of *P. vivax* and *P. falciparum* infections with the antigen derived from the cultures of either parasite, but the

reaction between a given serum and its homologous antigen is the more intense. Antigens made from normal blood cells, or from cultures of other protozoa, do not give reactions with malarious blood, nor does the malaria antigen react with sera from cases of syphilis, anaemia and other diseases. The reaction is strongest during the apyrexia after a paroxysm; it diminishes as time goes on until it completely disappears.

W. F.

SINTON (J. A.) & KEHAR (N. D.). **Changes in the Amount of Blood Sugar in Malaria.**—*Records of the Malaria Survey of India.* 1931. June. Vol. 2. No. 2. pp. 287–304. With 4 text figs. [39 refs.]

In 55 cases of benign tertian the blood sugar averaged 0·067 before the fever, 0·124 during the fever and 0·075 after the fever. In two cases of subtertian, the blood sugar rose to 0·206, during pyrexia, in one; and to 0·140 in the other. [See GREEN, this *Bulletin*, Vol. 27, p. 194.] The authors conclude that the rise is due to increased glycogenolysis similar to that which occurs in protein shock and that activity of the adrenals is responsible.

W. F.

JOHNS (F. M.). **Influence of Dextrose and of Low Temperatures on Preservation, Transportation and Viability of Malaria Parasites.**—*Proc. Soc. Experim. Biol. & Med.* 1931. Apr. Vol. 28. No. 7. pp. 743–745. [1 ref.] [Med. School, Tulane Univ. of Louisiana, New Orleans.]

Experiments were made with defibrinated blood from patients who had been inoculated with malaria. Concentrations of dextrose ranging from 0·5 to 2 per cent. were employed, and the blood was kept at temperatures ranging from —5° to +15° C. The parasites survived longest in blood containing 1 per cent. of dextrose, and kept at 0° C. The routine method adopted is: run the blood from the collecting syringe into a tube marked for 10 cc., containing 0·2 cc. of a 50 per cent. solution of dextrose. Defibrinate by stirring for 10 minutes with a roughened glass rod. Transfer to rubber-stoppered vaccine bottles of 5 cc. capacity and store in crushed ice. For transportation, pack in a thermos flask filled with ice and water.

The author concludes that "the infectivity of such blood up to 8 days is fully equal to direct transfer of blood from patient to patient." In two cases inoculated with blood 16–18 days old, one was infected, the other was not.

W. F.

AKASHI (Kazuyoshi). Ueber den Kochsalz-Gehalt des Blutes bei Malaria. [**Salt Content of the Blood in Malaria.**]—*Taiwan Igakkai Zasshi* (*Jl. Med. Assoc. Formosa*). 1931. May. Vol. 30. No. 5. [In Japanese. German summary p. 36.] [Govt. Hosp., Tainan, Formosa.]

The salt content of the blood in 15 healthy men and the same number of females was determined. The content of the blood serum and corpuscles was the same in both, but the salt content of the whole blood was much higher in females than in males, which the author attributes to the fact that the volume of corpuscles is smaller in women than in men. He then investigated 7 cases of malignant tertian, 6 cases of benign tertian and 2 cases of inoculated malaria. In all cases he found that the blood serum, corpuscles and whole blood showed a lowered salt content, but after treatment with quinine the content returned to normal. Reduction of salt content of the blood was not confined to malaria but occurred also in other febrile diseases.

E. D. W. Greig.

SINGH (Jamiat). **Urobilinuria and its Importance in Malaria.**—*Indian Med. Gaz.* 1931. May. Vol. 66. No. 5. pp. 241–244. [2 refs.] [King Edward Med. College, Lahore.]

This is an additional report on the author's investigations (see above, p. 141). Bilirubin is made from haemoglobin by the cells of the reticulo-endothelial system. The bilirubin is converted into stercobilin by micro-organisms in the large intestine. Some of the stercobilin is re-absorbed into the circulation and carried to the liver. If there is more stercobilin than can be dealt with there, some of it passes out through the kidneys as urobilinogen. In health, there is very little urobilinogen in the urine. Excess may be due to excessive blood destruction, in malaria and pernicious anaemia for example, or to the absorption of blood products from collections of blood within the body, such as cerebral haemorrhages or resolving lobar pneumonia. A strongly positive urobilin test with 8,000 to 30,000 units per 100 cc. of urine affords strong presumptive evidence of malaria. There is often a weak positive reaction—a pink colour—in lobar pneumonia, the urobilin units ranging from 4,000 to 8,000 in 100 cc. For quantitative estimation take 10 cc. of urine, add one drop of Lugol's iodine solution and 10 cc. of saturated zinc acetate in absolute alcohol; filter and compare with a standard solution of 1 mgm. of acriflavine in 30,000 cc. of distilled water, representing 1 unit (equivalent to 1 mgm. of urobilin dissolved in 950 cc. of the standard diluent). The comparison should be made in a Cole's comparator.

W. F.

PAWAN (J. L.). **Feulgen Nucleal Reaction and the Malarial Nucleus.**—*Ann. Trop. Med. & Parasit.* 1931. Aug. 13. Vol. 25. No. 2. pp. 185–187. [8 refs.]

The author summarizes his investigation as follows :—

"The modification of the Feulgen Nucleal reaction* applied here indicates :—

"1. That thymonucleic acid is absent in the composition of the nucleus of certain forms of human plasmodia.

"2. That the nuclei of these forms of plasmodia differ chemically from the nuclei of the protozoa studied by Robertson, viz., *Trypanosoma raiae*, *Bodo caudatus* and *Heteromita*."

W. F.

CLYDE (D.). **Report on the Control of Malaria during the Sarda Canal Construction (1920–1929).**—*Records of the Malaria Survey of India.* 1931. Mar. Vol. 2. No. 1. pp. 49–110. With 6 graphs, 1 map & 13 figs. on 8 plates. [2 refs.]

The Sarda Canal scheme will irrigate the districts of Carcilly, Shahjehanpur and Pilibhit as well as Oudh. The canal system is 4,000 miles long and is thus the largest in the world; it waters seven million acres of land, an area equal to more than a fifth of the whole of England. A rise in malaria, similar to that which followed the

* The Feulgen microchemical nucleal reaction is applied to the study of nuclear structure and in particular, nuclear chromatin. In a protozoal nucleus two groups of nucleic acid have been described, of which one is thymonucleic acid. The Feulgen reaction is indicative of the presence of nucleoprotein in the form of thymonucleic acid.

opening of the Ganges canal in 1854, has been guarded against by providing drainage cuts and prohibiting irrigation in all areas where the subsoil water is already high, and 1,800 miles of drainage canals have been laid down. This report deals mainly with the work carried out at the head works of the canal in the notorious Terai, where the spleen rate averages 75 per cent. The labour was imported by contractors, and the coolies were not under official control. The bad name of the district made it difficult to get them, and if they were pricked for blood examination, or were urged too strongly to take quinine, they absconded and there was trouble with the contractors. Construction work was suspended every year in May, at the beginning of the rains, and commenced again in November. During the intervals, the anti-malaria drains were washed away by the floods and the wreckage was buried in the undergrowth and weeds which sprang up. The coolies lived in grass huts and slept on the floor. Their diet was deficient in quality and also in the quantity of fats. Screening was not carried out ; without electricity and fans, screened houses in the United Provinces' Terai would be unbearable. Mosquito nets were used only by the more educated officers. The value of mosquito-proofed barracks for uneducated native labour is problematical ; the author believes that with regular fumigation and efficient inspection, properly proofed barracks would have been better than grass huts. Anopheles bred everywhere and they included practically every species known in the United Provinces. It is interesting that there were no culicines when the work was begun, though a few appeared when the jungle was felled and the camps built. The autumn malaria was probably due to *A. listoni* and the spring malaria due to *A. maculatus* and *A. culicifacies*. It is surprising that in the many thousands of wild anopheles examined no oöcysts or sporozoites were discovered.

Owing to the difficulties encountered, almost all the known direct and indirect anti-malaria measures were undertaken at one time or another. Elephant-grass jungle had to be cleared even after Paris green was used, not only to destroy the adult mosquitoes in it, but because the swamps under it were breeding profusely. This clearing provided suitable conditions for open breeders and in some places anophelines appeared where none had been before. Much draining was done, and oiling was carried out from 1921 to 1926 when Paris green was substituted. After 1926, efforts were concentrated upon Paris green dusting and fumigation in conjunction with treatment. The powder had no bad effect upon the larvivorous fish with which the area abounds. " It is extremely doubtful, in the United Provinces Terai at least, if larvicidal fish can be considered as of any use whatever." The following was used for fumigation of labourers' huts, out-houses, etc.—equal parts of sulphur and powdered waste tobacco leaves and stems ; half a pound burnt quickly per 1,000 feet of space. For the houses of officials a spray was used composed of carbon tetrachloride 1 per cent. and creosote 2 per cent. in kerosene. In the first years of the works, before anti-malaria measures were started, work had to be abandoned because 96 per cent. of the labour was down with malaria and the contractors refused to continue. All that is now claimed is that, as the result of the measures which were taken, malaria was controlled to such an extent that it became possible to begin again and to carry on the work to completion.

W. F.

NORRIS (Benjamin). **Malarial Control at Fort Stotsenburg.**—*Milit. Surgeon.* 1931. Apr. Vol. 68. No. 4. pp. 455–464.

Colonel CRAIG referred to Stotsenburg, in 1905, as "the malarial pest-hole of the army." Since 1925 a more vigorous anti-mosquito campaign has been carried on, and since 1928 carriers have been sought out and treated. The results are as follows :—471 cases of malaria in 1923 ; 415 in 1924 ; 232 in 1925 ; 136 in 1926 ; 141 in 1927 ; 140 in 1928 ; 46 in 1929 ; 10 in first half of 1930.

Drainage, oiling and dusting with Paris green are the antilarval methods adopted. Barracks and stores are sprayed with kerosene, and adult mosquitoes are killed. Soldiers are required to sleep under mosquito nets. Malaria patients are not discharged from hospital until their blood is free from parasites, and afterwards they have to visit the hospital daily for a three-months' course of quinine. Men on duty outside the controlled area are given prophylactic quinine. When a married soldier is admitted, his family is visited and, if necessary, treated. Plasmoquine has been in use since the end of 1928 ; a fourteen day course is administered to all carriers. The anti-mosquito work is carried out by nine civilian labourers and 22 soldiers. There is an annual anti-malaria fund of $2,500.

W. F.

i. LE PRINCE (J. A.). **Cost of Malaria Control.**—*Amer. Jl. Public Health.* 1931. Apr. Vol. 21. No. 4. pp. 378–381.

ii. FULLERTON (Howard R.). **Screening and Mosquito Proofing as Elements in Malaria Control.**—*Ibid.* pp. 382–389. [Tennessee State Dept. of Public Health, Nashville, Tenn.]

i. The cost of antilarval measures in 70 towns in ten southern states was between 70 and 80 cents a head for the first year and from 20 to 30 cents afterwards. This included ditching and larvicides. In the tropics, where mosquitoes are present all the year round the cost is higher ; in Panama, it was one cent a day for each person, or $3·65 in the year, exclusive of screening.

Millions of dollars are being spent in the southern states for the prevention of malaria by the great hydro-electric power-companies. Some of the lakes impounded for these purposes are hundreds of miles in circumference. Paris green will make the work cheaper ; but unfortunately it does not destroy culicines. During 1927, the Red Cross and the U.S. Public Health Service screened about 8,000 "farm-tenant homes" in the Mississippi River area at an average cost of $10 a house. The screening lasts about 5 years.

ii. "The local health departments do not do malaria control work exclusively, but carry it along as one of their major activities in their general health work." Screening alone does not make a Tennessee labourer's house mosquito-proof, because there are so many holes in the floor and the walls. This difficulty has been overcome by employing the tough heavy paper which is used for wrapping up tea samples. Many tons of this paper was obtained without charge from a cotton exchange. In 1928, the landowners and county officials of Lake County formed an anti-malaria committee. They provided a small capital, obtained a factory free of charge, and started to manufacture screen-doors and windows, with the result that 90 per cent. of the county has

been screened. In Gibson County a meeting of dealers was held and $2·25 agreed upon as the price of a screened door, only 16 mesh wire to be used.

W. F.

WALCH (E. W.), VAN BREEMEN (M. L.) & REYNTJES (E. J.). **The Sanitation of the Saltwater Fishponds of Batavia (a Contribution towards the "Hygienic" Exploitation of the Bandeng-Ponds).**—*Meded. Dienst d. Volksgezondheid in Nederl.-Indië.* 1930. Vol. 19. Pt. 3. pp. 400–430. With 4 figs. & 8 plates. [Refs. in footnotes.]

The work described in this paper illustrates the value of a special Technical Sanitary Department of the Medical and Sanitary Service. It also illustrates the value of co-operation between the various branches of a Government Service. Here all helped: the fisheries department, the war department (with photographs from the air), the agricultural, the museums, etc. The article deals with the application of the Pasoeran method of exploitation to the fish-ponds of Batavia. (See DE VOGEL above, p. 110, and Walch and SCHUURMAN, 1930, Vol. 27, p. 640.) The results were most satisfactory. Within a few months the flora of the ponds was changed, and anopheles were banished although they continued to breed in the untreated fishponds alongside. This was accomplished without injury to the fish breeding industry. The authors encountered much opposition from the fish breeders and are to be congratulated on the determination, energy and tact with which they carried through their work to the satisfaction of all concerned.

W. F.

MALAYAN MEDICAL JOURNAL. 1930. Sept. Vol. 5. No. 3. pp. 108–109.—**Silt-Pitting on Rubber Estates in Relation to Mosquito Breeding and Malaria.**

To stop surface wash of water and erosion on undulating or hilly land during or after heavy rainfall and to assist natural percolation through the soil, contour drains are made; preferably by banking the soil, with or without silt pits. In heavy soil (impermeable or semi-impermeable) the contour drains should run to a common outlet, the discharge being through stops which allow the water to trickle away and prevent sudden rushes; rubble, brushwood, etc., may be employed in construction. Drainage systems on this principle for such special cases have already been reported as functioning successfully in Malaya.

Since silt-pitting became a widespread practice in agricultural land in the Malaya Peninsula, the larvae of dangerous anopheles, notably *Anopheles maculatus*, have been found by many observers and on many occasions in water standing in silt-pits on hillsides and on relatively flat land. The tendency for *A. maculatus* to breed in such pits undoubtedly becomes greater where other natural waters are regularly and efficiently oiled.

Since silt pits or drains are necessary in old rubber plantations, measures should be adopted within forty chains of dwellings for the prevention of anopheline mosquito breeding. Such possible measures are:—(*a*) Measures to prevent retention of excess water. Although pits are used to conserve water in most cases, other cases require removal by drainage. (*b*) The use of larvicides. The easiest method, the report states, is to apply fuel oil with a very small mop, and this

need be done only once a fortnight if the water be stagnant. Such oiling of all waters retained in pits in a residential area is being performed at present by a number of estates as part of the regular anti-malarial work. (*c*) In flat lands trenches should be continuous without stops. In residential areas it is recommended that contour drains be made by banking with surface soil. Such systems should always be adopted in the anti-malarial areas in new clearings, in place of pitting or digging trenches, unless such special soil conditions exist as would make pitting indispensable.

H. Home.

BENNETT (H. Leigh). **Anti-Malarial Drainage.**—*Kenya & East African Med. Jl.* 1930. Oct. Vol. 7. No. 7. pp. 190–198.

In discussing the question of swamp drainage the author points out the value of contour drains to cut off seepage. In a drainage system it is in certain cases necessary, to prevent anopheline breeding, to arrange to dry out a part of the drainage in rotation by shutting off a section and discharging through the remaining drainage. Earth channels are difficult to maintain in shape and grade and for any permanent anti-malarial work a stone, brick, or concrete invert and bottom section is necessary. Especially is this the case where the discharge from dwellings is taken. The sides of the channel should be sloped and firmly turfed and storm water can then be discharged without erosion following.

Subsoil drainage is often of value not only for the drainage of valley bottoms, etc., but also for flat areas to be occupied by dwellings. The author instances the case of a site at Kuala Lumpur, which had been turned down as too water-logged for even a night-soil dump, was drained by this method and is now a flourishing settlement. In the case of the type of swamp in which there is no natural discharge because of the land contours and of which the bottom is sealed by an impermeable stratum, successful work has been carried out through vertical drainage. A well is sunk through the impermeable stratum to lower permeable beds and from this well radial arms of subsoil piping should be laid at say 3 foot depth. The well coping should be carried up about 2 feet above ground level to cut off silt which may be washed in by storm water. The value of tree planting for the drying-out of swamp areas should not be overlooked. Eucalyptus (*robusta* and *soligna*) transpire very heavily and have the effect of drying-out large swamps.

The author mentions several farms in Eldoret and Kitale districts on which there are swamps that the owners are not attempting to cultivate because of the expense of drainage. These swamps could easily be dried out completely in about five years by planting trees, the cost of which would not exceed £3 per acre.

H. Home.

BARROWMAN (Barclay). **Anti-Malarial Oils and their Specification.**—*Malayan Med. Jl.* 1931. Mar. Vol. 6. No. 1. pp. 7–11. With 7 charts in text.

Five rubber estates used an oil composed of kerosene, solar oil and crude oil, which was purchased already mixed. In 1928 there were many cases of malaria, and the larvae of *A. maculatus* were found living in the water which had been oiled with this stock mixture. Adjoining

estates which prepared their own oil mixtures did not suffer in the same way, and when the five estates in question followed their example, mosquito breeding was checked and the monthly incidence of malaria fell from 3·3 to 0·2 per cent. A mixture composed of kerosene 4 parts, solar oil 20 parts, and crude oil 40 parts was found the most efficient. The value of an oil is usually estimated by its power to kill mosquito larvae when it is added to water containing them. But in practice, the most toxic oil is not always the most successful in controlling malaria ; its spreading and lasting qualities must be taken into account, for the main purpose of an anti-mosquito oil is to make the water repulsive to the adult insects and so prevent their laying their eggs in it.

W. F.

BOSE (K.). **Application and Use of Larvicides.**—*Indian Med. Gaz.* 1931. Aug. Vol. 66. No. 8. pp. 436–440. With 2 text figs. [3 refs.]

The Birnagar Palli Mandali (see this *Bulletin*, Vol. 26, p. 909) has now adopted the use of Paris green, distributed by means of American rotary blowers, as an antilarval measure. Brick dust from old ruins is a satisfactory diluent. Where it formerly took a gang of men 8 days to complete a round of spraying with oil, it is now possible to " green " the whole area in 2½ hours. Moreover, no clearing of weeds is necessary when Paris green is used, and this is a great saving. " Wider use of this larvicide has hitherto been hampered mainly for lack of information in India about the suitable type of blowers." [This has been remedied by the publication of Major COVELL's book, see below, p. 1035.

W. F.

BOGOPOLSKY (M.). [**Prophylactic Quininization of Chronic Malaria Cases.**]—*Trop. Med. & Vet.* Moscow. 1930. Vol. 8. No. 6–7. pp. 11–13. [In Russian.]

Systematic prophylactic administration of quinine for chronic cases of malaria has been carried out in Kiev, at the Regional Malaria Station from 1925 to 1927. The course was as follows : from January to July, 0·5 gm. quinine hydrochloride every 7th and 8th days (=20 gm. in 6 months) ; from July onwards—the same dose once a week. The results appeared to be satisfactory ; in 1925 no relapses were observed, while in 1926 out of 771 cases, 34 (4·4 p.c.) relapsed ; in 1927 there were 19 (3·3 p.c.) relapses among the 572 chronic cases treated.

C. A. Hoare.

JAMES (J. F.). **Quinine Prophylaxis and Other Notes on Malaria.**—*Indian Med. Gaz.* 1931. Aug. Vol. 66. No. 8. pp. 440–443. With 2 text figs. [Indian Military Hosp., Nowshera.]

This paper deals with the administrative side of prophylaxis with quinine among soldiers. In a series of 6,000 men to whom quinine was given, two cases of quinine idiosyncrasy occurred. The Boerner skin reaction was positive with quinine in both cases. The symptoms were, puffiness of the face, erythema, vomiting, diarrhoea, itching and urticaria. An injection of adrenalin gave relief in a few minutes. Both patients could take quinidine or plasmoquine with impunity.

W. F.

Gosio (Renato). Contributo clinico allo studio delle gravi anemie insorgenti nelle malariche in gravidanza. (**Clinical Contribution to the Study of the Severe Malarial Anaemias in Pregnancy.**)—*Riv. di Malariologia.* 1931. Jan.-Feb. Vol. 10. No. 1. pp. 52–85. With 6 text figs. [16 refs.] English summary p. 158. [Roy. Inst. of Clin. Med., Univ., Rome.]

This paper is based upon a study of 15 patients. Seven of these cases in which the degree of anaemia was severe are fully detailed [but in five of them parasites could not be found].

H. H. S.

Onnizew (P.). Untersuchung der Retraktion des Blutkoagulums bei Malariakranken. [**Retraction of the Blood Clot in Malaria.**]—*Odessaer Med. Ztschr.* 1930. Vol. 5. No. 3–5. pp. 255–257. [9 refs.] [In Russian. German summary (5 lines).]

A number of observations conducted with the object of determining the factors influencing the appearance of haemorrhagic diathesis in malaria.

C. A. Hoare.

Advier. Deux cas d'infection malarique sans manifestation clinique.—*Marseille-Méd.* 1931. Mar. 25. Vol. 68. No. 9. pp. 420–423. [7 refs.]

Banerji (Kali Gati). Atypical Cases of Malaria as seen in the Recent Outbreak in Birbhum.—*Calcutta Med. Jl.* 1931. June. Vol. 25. No. 12. pp. 454–463.

v. Berkesy (L.). Ueber die Entstehung der Malariainfektionen im Vorfrühling.—*Wien. Klin. Woch.* 1931. Aug. 28. Vol. 44. No. 35. pp. 1110–1112. [Med. Clinic, Franz Joseph Univ., Szegedin, Hungary.]

Bey (Sami). Les difficultés du diagnostic clinique de la malaria.—*Rev. Prat. Malad. des Pays Chauds.* 1930. Dec. Year 9. Vol. 10. No. 12. pp. 570–572.

Blanc (F.) & Goiran (E.). A propos d'un cas de paludisme autochtone.—*Marseille-Méd.* 1931. Mar. 25. Vol. 68. No. 9. pp. 417–419. With 1 chart in text.

Butt (N. M.). A Simple and Inexpensive Portable Screener for Use with Paris Green Diluents.—*Records of the Malaria Survey of India.* 1931. June. Vol. 2. No. 2. pp. 333–335. With 1 text fig.

Castillon (T. J. E. L.). La lutte antipaludique dans les Etats sous mandat français : Alexandrette (1919-1929).—*Arch. Méd. et Pharm. Milit.* 1931. Apr. Vol. 94. No. 4. pp. 541–574. With 2 maps, 1 chart & 4 figs. [Refs. in footnotes.]

Collier (W. A.) & Warstadt (A.). Untersuchungen ueber die Wirkung eines neuen Malariamittels bei der Malariainfektion des Kanarienvogels und des Menschen.—*Klin. Woch.* 1931. May 23. Vol. 10. No. 21. pp. 987–988. [Robert Koch Inst. for Infectious Diseases, Berlin.]

Costantini (Henri). Paludisme et chirurgie.—*Riv. di Malariologia.* 1930. Nov.-Dec. Vol. 9. No. 6. pp. 759–776. [41 refs.] [Surg. Clinic, Mixed Faculty of Med. & Pharm., Algiers.]

Cuboni (E.) & Milani (C.). Inoculazione della malaria nell'uomo ed immunità antimalarica.—*Boll. Istituto Sieroterap. Milanese.* 1931. May. Vol. 10. No. 5. pp. 225–236. With 3 text figs. [14 refs.]

Decourt (Philippe). Index splénique et rapport splénique d'endemicité.—*Rev. Méd. et Hyg. Trop.* 1931. May-June. Vol. 23. No. 3. pp. 141–145. [2 refs.]

da Fonseca (Olympio), Jr. Bases scientificas e realisação pratica da prophylaxia do impaludismo.—*Rev. Med.-Cirurg. do Brasil.* 1931. Mar. Vol. 39. No. 3. pp. 65–96.

Geiger (J. C.) & Gray (J. P.). Note on an Outbreak of Malaria in a Railroad Camp, Rawson Switch, Calif.—*Public Health Rep.* 1931. Mar. 6. Vol. 46. No. 10. pp. 516–518.

GUY (Roger). Note sur quelques documents relatifs au paludisme dans le Haut-Mékong.—*Bull. Soc. Méd.-Chirurg. Indochine.* 1931. May. Vol. 9. No. 5. pp. 305–338. With 5 graphs. [4 refs.]

HOPKINS (H. O.). Pancreatic Efficiency Tests in Malaria.—*Bull. Inst. Med. Res. Federated Malay States.* 1929. No. 1. 8 pp. [20 refs.]

KLIGLER (I. J.). Organisation du controle malarique en Palestine.—*Rev. Prat. Malad. des Pays Chauds.* 1930. Dec. Year 9. Vol. 10. No. 12. pp. 564, 566–569.

LEBLANC (L.). Le problème de la malaria à la Fomulac.—*Ann. Soc. Belge de Méd. Trop.* 1931. Mar. 31. Vol. 11. No. 1. pp. 47–57. With 1 chart & 1 map.

MORIN (Henry G. S.). Sur l'organisation du service antipaludique des Instituts Pasteur d'Indochine.—*Bull. Soc. Méd.-Chirurg. Indochine.* 1931. May. Vol. 9. No. 5. pp. 271–280. [Pasteur Inst., Hanoi.]

MORISHITA (Kaoru), NAMIKAWA (Hiroshi), MATSUURA (Tamotsu) & TANIKAWA (Kunishizu). Report on the Malaria Epidemic in Uzanto and its Control, with Special Reference to the Effect of all Quininization.—*Taiwan Igakkai Zasshi* (*Jl. Med. Assoc. Formosa*). 1931. July. Vol. 30. No. 7 (316). [In Japanese. English summary pp. 53–54.] [Govt. Research Inst., Formosa.]

O'FLYNN (J. A.). Control of Malaria at H.M. Naval Base, Singapore (1925–1927).—*Jl. State Med.* 1931. Aug. Vol. 39. No. 8. pp. 446–452.

ROCHA (Francisco). Malaria Control at "Mina de S. Domingos."—*Arquivos Inst. Bact. Camara Pestana.* 1930. Vol. 6. No. 2. pp. 123–137. With 1 folding plan, 1 folding map & 2 folding charts. [17 refs.] [Camara Pestana Inst., Lisbon.]

ROSS (G. A. Park). The Routine Treatment and Prophylaxis of Malarial Fever.—*Jl. Med. Assoc. South Africa.* 1931. Mar. 14. Vol. 5. No. 5. pp. 146–148. [7 refs.]

SARKAR (Sarasi Lal). A Malaria Survey in Noakhali District, Bengal.—*Indian Med. Gaz.* 1931. June. Vol. 66. No. 6. pp. 322–326. With 1 map in text.

SCHORLEMMER (R.). Kann eine vorausgegangene Malaria mit Darmstörungen Jahre hindurch ursächlich für periodisch einsetzende Reizzustände und Geschwürsbildung im Dickdarm in Frage kommen? Ein dem Reichs-Versorgungsgericht auf Ansuchen erstattetes Gutachten.—*Arch. f. Verdauungs Krankht.* 1931. June. Vol. 49. No. 5–6. pp. 315–332.

SINTON (J. A.). The Treatment of the Malarial Fevers.—*Malayan Med. Jl.* 1931. June. Vol. 6. No. 2. pp. 33–37.

SPENCER (H. H.). The Malarial Syndromes.—*Jl. Med. Assoc. South Africa.* 1931. May 9. Vol. 5. No. 9. pp. 275–278.

TANON (L.), CAMBESSÉDÈS (H.) & DECOURT (Ph.). Enquête sur le traitement du paludisme.—*Rev. Méd. et Hyg. Trop.* 1931. Mar.-Apr. Vol. 23. No. 2. pp. 65–85.

REVIEWS AND NOTICES

Proceedings of the Celebration of the Three Hundredth Anniversary of the First Recognized Use of Cinchona. Held at the Missouri Botanical Gardens, St. Louis, October 31-November 1, 1930.—258 pp. With 7 plates. 1931. St. Louis, Mo., U.S.A.

The Missouri Botanical Garden where this congress was held has a large lecture hall, a very fine library, and a garden of 1,600 acres which contains the greatest collection of orchids in the world. The congress was attended by many whose names are well known in connexion with quinine; among them were Dr. KERBOSCH, director of the government cinchona estates and experimental station in Java, whence comes 97 per cent. of the world's supply of bark; Dr. VAN LINGE, president of the Nederlandsche Kininefabriek and head of the so-called Dutch quinine monopoly; Dr. Karl MERCK, president of E. Merck of Darmstadt; Mr. BESANT, director of the Botanical Gardens in Glasgow; Messrs. G. D. and J. ROSENGARTEN of Philadelphia, whose firm produced quinine in 1823, three years after it was first isolated by PELLETIER and CAVENTOU, and many other well-known American quinologists. It is not surprising that, with such a company, the published Proceedings are of very great interest. Dr. KERBOSCH's paper on the history of cinchona in Java is too important to be dealt with in a short review, and a separate summary of it will be found on page 817 of this *Bulletin*. The debt of gratitude which the world owes to the Dutch for the scientific cultivation of the cinchona tree was acknowledged at the banquet which terminated the congress, by the playing of the Dutch national anthem, while all the guests stood in silence. One of the guests at this banquet told how W. H. PERKIN was attempting to synthesize quinine in 1852, when he made, by accident, mauve, the first of the synthetic dyes, and how von HOFMANN came over to England, learned Perkin's method of work, and went back to Germany to establish the famous aniline dye industry.

Dr. Leo SUPPAN contributed a scholarly paper entitled "Three Centuries of Cinchona," which occupies 120 pages of the Proceedings and contains 150 references. It is probably the most complete and concise history of cinchona which has been written in English. When the drug was first introduced into Europe it met with great opposition; the Galenists were bitterly opposed to it, and the Protestants, who scented a Romanist plot, announced that the bark was an insidious poison which the Jesuits had brought to Europe to exterminate them.

The early immigrants to the Mississippi Valley suffered terribly from malaria, and the political enemies of Thomas Jefferson protested that in the Louisiana Purchase the United States had acquired a swamp unfit for habitation. To-day it is the home of millions, and Dr. Robert TERRY in his address to the congress gave an interesting account of Dr. John SAPPINGTON, a pioneer in the use of quinine, whose work did so much to make the colonization of the Valley possible in the early days of the nineteenth century.

Dr. R. ROSENGARTEN and Dr. T. SOLLMANN dealt with the alkaloids of cinchona bark, and it is interesting to note that chinoidin may prove of great use in the moth-proofing of furs and fabrics. Dr. SOLLMANN pointed out that though EHRLICH invented the name chemotherapy, the phenomenon was recognized before his day, and was exemplified by cinchona. Its mode of action is still, as he said, unknown. Direct parasiticidal action does not account for it; blood incubated for 24 hours with 1 in 10,000 quinine is still infective, and the concentration of quinine in the circulating blood cannot be maintained nearly as high as this. The assumption of toxic transformation in the body is unsupported by chemistry and the effect of quinine is too prompt to give opportunity for the manu-

facture of antibodies. Dr. Kenneth MAXCY pointed out, in the paper which he contributed on the rôle of quinine in malaria, that though quinine may inhibit the movements and growth of malaria parasites *in vitro*, such experiments are inconclusive, for there are many substances which would act in the same way though they are totally without effect in the treatment of malaria.

W. Fletcher.

RÖDER (Richard). **Die anthropogeographische Bedeutung der Malaria.** [Geographical Distribution of Malaria.]—*Janus.* 1930. Jan., Feb., Mar., Apr. & May-June. Vol. 34. Nos. 1, 2, 3, 4, 5 & 6. pp. 1–29; 38–64; 71–96; 121–128; 142–176. With 5 text figs. & 1 folding map. [Refs. in footnotes.]

This essay on the significance of the anthropogeography of malaria has been compiled by Dr. Röder with the help of Professors Dr. Karl SAPPER and Dr. Georg STICKER. It begins with a short description of malaria and of the plasmodia that cause its separate types. Then follows a long section dealing with the geographical distribution over the different continents in time and space. Figures and charts are given to show the progress of the disease for such periods as can be connected with facts and statistics. In England and northern Europe malaria is not at the present day of serious importance; but in the south and south-east its influence on life is still serious. Of the Americas much the same may be said, comparative freedom in the north, greater frequency in Southern, Central and S. America. It is in the wide belt of tropical and subtropical regions that the relation between malaria and anthropology is most serious and important. The statistics of morbidity and mortality only tell part of the story. There are thousands of cases in Asia, Africa, etc., never showing a high rate of "fever" but practically all their lives subject to malarial infection, and this applies to children as well as adults. The weakness and anaemia produced lessens the working and productive power of individuals, reduces the vigour of the population and has an evil influence on the history of nations. There is also less chance of healthy colonization in areas strongly infected and colonists or excess population can only make use of such areas when the scourge of malaria has been reduced as far as is possible—*e.g.*, Panama. It would appear that although malaria confers no lasting immunity on the sufferers there may be, in time, developed a certain racial resistance though not of great value. The moving of infected people into non-infected areas is a danger to be considered since if suitable anopheles carriers are present the disease may spread. Examples of this were seen in France and England during and after the war of 1914–18. The authors devote some pages to the geographical distribution of the Anopheles. The conditions for the spread of malaria being what they are it becomes a matter of anthropological importance that swampy areas should be drained and cultivated; but this is not always possible since crops such as rice, jute and flax require large quantities of water and encourage mosquitoes. Houses must be properly screened to prevent mosquitoes reaching infected or healthy persons and quinine must be available for treatment. The most difficult and expensive work is the destruction of larval and adult mosquitoes. The abolition of malaria in any area is a boon to mankind. Much has been done during the last fifty years to eradicate the disease in various parts of the world; but far more remains to be done. Dr. Röder writes as a conclusion: "Man hat die Bedeutung der Malaria für das Leben und das Glück der Völker stark übertrieben"!

J. H. Tull Walsh.

COVELL (Gordon) [M.D. (Lond.), D.P.H., D.T.M. & H. (Eng.), Major, Indian Medical Service, Assistant Director, Malaria Survey of India]. **Malaria Control by Anti-Mosquito Measures.**—pp. ix+148. With 13 text figs. & 2 plates. Calcutta & Simla: Thacker, Spink & Co., Ltd. London: W. Thacker & Co., 2 Creed Lane, E.C. [7s. 6d.]

The papers which have been written about anti-mosquito measures are so numerous, and they have been published in so many different journals, that it has become difficult to keep in touch with them. Major Covell has made a successful attempt to remedy this by collating the information which they contain, and in this book he gives a concise account of the methods advocated. Part I deals with protection against mosquito bites by means of nets, culicifuges, and screening. Part II deals with the destruction of adult mosquitoes by traps, fumigation, sprays, and the likel Part III, which is the largest section, describes the many methods of larva. control; it is divided into two subsections, (*a*) giving details of the different methods, and (*b*) indicating the special means which should be employed in special conditions; e.g., the respective measures which should be adopted in dealing with streams and with swamps. It is impossible, as the author says, to deal very fully with the subject of drainage in a book of this kind, but the various methods of oiling and of distributing Paris green have been treated in considerable detail. A list of the places where anti-mosquito apparatus can be purchased is given in an appendix, together with the prices of the various articles; as the book is intended primarily for use in India, the firms mentioned are nearly all in that country and the prices are given in rupees. There is a useful bibliography containing 570 references. Among the many control measures mentioned, some are of doubtful value; the author gives his own opinion of them in a number of cases, but, in others, the reader has to make his choice unaided. For instance, on page 76 one finds "cactus paste has been used in North Africa by the French. . . . This is spread on the surface of stagnant water and forms a layer which prevents the larvae coming to the top to breathe. It is claimed that this paste can act for weeks, months, or even years." It is only on turning to the Bibliography that one finds that this information comes from a paper published 25 years ago. The section on Paris green contains much practical information and should be of great use in India.

W. Fletcher.

GARDNER (Gérard) [Docteur de l'Université de Paris, Chargé de cours à la Faculté des sciences de l'Université de Montréal et à l'Ecole des Hautes Etudes commerciales]. **Recherches sur les spirochétidés dans le district de Montréal.** [Researches on the Spirochaetes of Montreal.]—88 pp. With 7 plates (2 double). 1930. Paris: Editions médicales. 7 rue de Valois (1er).

The first part of this thesis, comprising a third of the volume, is devoted to a discussion of the general characteristics of spirochaetes, including nomenclature. The author then proceeds to give records of his observations on spirochaetes in Canada, which are of interest for comparison with those from other parts of the world.

Fresh water from various sources was examined both directly after collection and also after standing with the addition of hay, peptone, sugar, etc. The results show that the Canadian waters are extremely poor in spirochaetes as compared with those of Europe. Sea water was also examined and contained various types resembling those recorded from other parts of the world. By the maceration of vegetation in sterile tap water a number of spirochaetes were obtained including *S. biflexa*, *S. pseudo-ictero-haemorrhagiae*, and in particular a distinct type referred to

as the " type de la Phléole " cultivated from timothy grass. This spirochaete resembles *S. plicatilis* but is very much shorter, a maximum of 15μ instead of lengths up to 500μ. Among other observations on the morphology of this spirochaete it is stated that the addition of 1-1000 oxazine causes the surface membrane and granulations to become distinct and also reveals the existence of an axial filament.

The intestines of a number of animals both vertebrate and invertebrate were examined with negative results, but spirochaetes were obtained by cultivation of the cud of a cow, and a number of rabbits were found to be infected with *S. cuniculi* and to harbour a spirochaete in the caecum.

A separate section is devoted to the problem of the occurrence of *S. icterohaemorrhagiae* in nature, and in common with practically all other observers, he found strains of free living leptospira to be non-pathogenic. Nevertheless after being cultivated for two or three years these free-living strains acquired certain characteristics of the pathogenic type, in particular, agglutinability by human convalescent serum from cases of Weil's disease.

Finally the author gives the results of exposing spirochaetes to the action of cold, cultures of *S. icterohaemorrhagiae* being kept at a temperature of approximately —15° C. The spirochaetes were found to gradually lose their motility and break up into granules, and subcultures of these forms were negative. It would seem that the comparative rarity of spirochaetes in Canadian waters may be attributed to the low winter temperatures which are probably fatal to most free living forms.

E. Hindle.

JORDAN (Edwin Oakes) [Chairman of the Department of Hygiene and Bacteriology, University of Chicago]. **Food Poisoning and Food-borne Infection.**—pp. xi+286. With 22 text figs. 1931. Chicago, Illinois: University of Chicago Press. London: Cambridge University Press, Fetter Lane. [11s. 6d.]

A review of this work (by Dr. W. G. Savage) will be found in the *Bulletin of Hygiene*, Vol. 6, p. 726. The reviewer writes—

" The volume may be recommended as a very readable and comprehensive account of the varieties of food poisoning."

We record with much regret the death on November 27 of SIR DAVID BRUCE. Sir David had been a member of the Honorary Managing Committee since the institution of the Tropical Diseases Bureau in 1912, and previously of the Sleeping Sickness Bureau (1908–12).

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The bracketed abbreviations after the page numbers indicate the subjects.
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Am. signifies Amoebiasis and Amoebic Dysentery.
Bb. ,, Beriberi and Epidemic Dropsy.
Bl. ,, Blackwater.
B.R. ,, Book Review.
Chl. ,, Cholera.
C.Bu. ,, Climatic Bubo and Lymphogranuloma Inguinale.
Der. ,, Tropical Dermatology.
Dys. ,, Dysentery (Bacillary and Unclassed).
Fev. ,, Fevers.
G.V. ,, Granuloma Venereum.
Hel. ,, Helminthiasis.
Hist. ,, Historical.
H.S. ,, Heat Stroke.
K.A. ,, Kala Azar.
Lab. ,, Laboratory Reports.
Lep. signifies Leprosy.
Lept. ,, Leptospirosis.
Mal. ,, Malaria.
Misc. ,, Miscellaneous.
Myc. ,, Tropical Mycology.
Oph. ,, Tropical Ophthalmology.
Pel. ,, Pellagra.
Pl. ,, Plague.
Rab. ,, Rabies.
R.B.F. ,, Rat-Bite Fever.
R.F. ,, Relapsing Fever and other Spirochaetoses.
Sp. ,, Sprue.
S.S. ,, Sleeping Sickness.
Und. ,, Undulant and Abortus Fever.
Y.F. ,, Yellow Fever.
Y. & S. ,, Yaws & Syphilis.
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—— & de Rook, H., 217 (Hel.)
Brumpt, E., 189, 193 (Hel.), 629 (Fev.)
—— & Chevallier, P., 670 (Hel.)
Brun, C., with Vaudremer & Sézary, 657 (Lep.)
Brunelli, P., (801) (Am.)
Brunsting, L. A., 934 (Y. & S.)
Brussin, A. M. & Kalajev, A. W., 919 (S.S.)
Bruxelles-Médical, 762 (Misc.)
Buchanan, G. & Roux, P., 796 (Dys.)
Buchanan, G. S., 883 (Chl.)
Buchanan, R. E. & Fulmer, E. I., 168 (B.R.)
Buchbinder, L., 295 (Y.F.)
de Buck, A., with Swellengrebel, 1018 (Mal.)
de Buen, S., 128, 591, 1008 (Mal.)
Bulletin de l'Office Internationale d'Hygiène Publique, 392 (Pl.)
Bunting, C. H., with Jones (692) (Hel.)
Burgess, A. S., 389 (Pl.)
Burgess, J. F. & Usher, B., 413 (Der.)
Burke, A. W. & Davis, N. C., 289 (Y.F.)
Burke-Gaffney, H. J. O'D., 837 (Misc.), 933 (Y. & S.)
Burkitt, 134 (Mal.)
Burnet, F. M., McKie, M. & Wood, I. J., 273 (Dys.)
Burowa, L. F. & Moltschanow, S. A., 154 (K.A.)
Bush, L. M., with Corke, 843 (Misc.)
Butler, C. S., 439, 931 (Y. & S.)
—— & Hernandez, V., 828 (Hist.)
Butt, N. M., (1031) (Mal.)
Buxton, P. A., 47 (Misc.), 533 (Z.)

C

Caballero, G. M., with Pardo-Castello, 653 (Lep.)
Cabu, F., with Schwetz & Baumann, 128 (Mal.)
Caffrey, P. J., 403 (Der.)
Caius, J. F., Mhaskar, K. S. & Isaacs, M., 68 (Misc.)
——, with ——, 68 (Misc.)
Calcutta, 778 (B.R.)
Caldwell, E. L., with Caldwell, F. C. & Davis, 222 (Hel.)
Caldwell, F. C., Caldwell, E. L. & Davis, G. E., 222 (Hel.)
Callens, J., 330 (Lep.)
Callerio, G., 232 (Hel.)
Camacho-Moya, J., with Thonnard-Neumann & Brewster, 402 (Der.)
Cambessédès, H., with Tanon & Decourt, (1032) (Mal.)
Cameron, T. W. M., 204 (Hel.)
Cameron, W. M., with Smyth, 102 (Fev.)
Caminopetros, J., with Blanc, 91, 618, 628 (Fev.), 153, 640 (K.A.), 1006 (Mal.)
Campos, E. de S., (925) (S.S.)
Campos, F. M. & Campos, P. S., 241 (Hel.)
Campos, N. de S., 325 (Lep.)
Campos, P. S., with Campos, F. M., 241 (Hel.)
Camps-Campins, J., with Pawan, 863 (Misc.)
Canaan, T., 648 (K.A.)
Candido, G., 745 (Rab.)
Cannavò, L., (636) (Fev.)
Cannon, P. R. & Taliaferro, W. H., 494 (Z.)
Caragianopoulos, G., with Pédaros & Valsamaki, 628 (Fev.)
Cardamatis, J. P., 486 (Z.)
Carley, P. S., 1019 (Mal.)
Carmody, E. P., 13 (Bl.)
Carneiro, H., 715 (Y.F.)
Caronia, G., 156 (K.A.)
Carpano, M., 485 (Z.)
Carpenter, G. D. H., 890 (S.S.)
Carpentier, G., 46 (Misc.)
Carranca Trujillo, R., (474) (Pel.)
Carrion, A. L., 397 (Der.)
Carroll, H. H., with Phelps, Smith, Washburn & Beagley, 215 (Hel.)
Carrosse, 669 (Hel.)
Carrosse, J., 188, 189 (Hel.)
Cartañá, P., 603 (Mal.)
Carter, H. F., 499 (Z.), 581, 582 (Mal.)
Carvalho, A. E., with Prado, 114 (Mal.)
Case, C. E., with Baumgartner, 663 (Sp.)
Casoni, T., 788 (Am.)
Castaneda, M. R. & Zinsser, H., 96 (Fev.)
Castellana, A., 785 (Am.)
Castellani, A., (280) (Dys.), 423 *bis* (Myc.), 429 (B.R.), 593 (Mal.), 776, (866) (Misc.)
Castello, P. & Mestre, J. J., 414 (Der.)
Casten, V., 67 (Misc.)
Castex, M. R., Poletti, R. A. & López García, A., (280) (Dys.)
Castillon, T. J. E. L., (1031) (Mal.)
Castles, R., with Havens, 229 (Hel.)
Catanei, A., 399 *bis* (Der.), 418 *bis* (Myc.)
——, with Sergent, Edm. & Sergent, Et., 592 (Mal.)
——, with ——, ——, Parrot, Foley & Senevet, 984 (Mal.)
——, with ——, ——, Trensz & Sergent, A., 1013 (Mal.)
——, with Sergent, Et., 599 (Mal.)
Cawadas, E., (147) (Mal.)
Cawston, F. G., 66 (Misc.), 182, 192, 193, (241 *bis*) (Hel.), (552) (Z.)
Cazamian, P., 886 (H.S.)
Cazanove, (147) (Mal.), 297 (R.F.), 381 (Pl.)
Cazanove, F., 93 (Fev.)

Celli, A., 126 (Mal.)
Celli-Fraentzel, A., 810, 814 (Hist.)
Ceppi, E., 226 (Hel.)
Cerqua, S., 214 (Hel.)
Cerruti, C., 88 (Und.)
Cerruti, C. F., 275 (Dys.)
Cesarando, U. & Giordano, M., (552) (Z.)
Ceylon, 499 (Z.), 610 (Mal.), 686 (Hel.)
Chacon, A. L., 163 (K.A.)
Chadwick, C. R., with Mills & Machattie, 158 (K.A.)
Chagas, E., 377 (S.S.)
Chaigneau, Y., 221 (Hel.)
Chalam, B. S., 578 (Mal.)
Chamberlain, W. P., 770 (Misc.)
Champagne, R., with Senevet, 182 (Hel.)
Chandler, A. C., Milliken, G. & Schuhardt, V. T., 217 (Hel.)
Chaniotis, N., with Lorando, 615 (Fev.)
Chantriot, 993 (Mal.)
Chao, S. S., with Reiner & Leonard, 359 (S.S.)
Charlier, M. & Charlier-Collon, N., 187 (Hel.)
Charlier-Collon, N., with Charlier, 187 (Hel.)
Chaskina-Munder, G., 275 *bis* (Dys.)
Chatterji, C. D., (147) (Mal.)
Chatterji, S. N., 652 (Lep.)
Chen, F. K., with Kurotchkin, 396 (Der.)
Chen, M. Y. & Anderson, H. H., 850 (Misc.)
——, —— & Leake, C. D., 851 (Misc.)
Cherefeddin, O., 141 (Mal.)
Cherian, P. V. & Vasudevan, A., 422 (Myc.)
Chesneau, P., 441 (Y. & S.)
—— & Tran-Van-Manh, (241) (Hel.)
Chesterman, C. C., 442 (Y. & S.), 857 (Misc.)
Chevalier, G., 159 (K.A.)
Chevallier, P., with Brumpt, 670 (Hel.)
Chewtchenko, F. I., with Chodoukine & Sophiew, 641 (K.A.)
China Medical Journal, 637, 639 (K.A.)
Chin-Kyu-Sui, Shimokawa, H. & Kamizawa, O., (866), (867) (Misc.)
Chitre, G. D., with Webster, 529 *bis* (Z.)
Chiyuto, S., 330 (Lep.)
Chodoukine, N. I., Sophiew, M. S. & Chewtchenko, F. I., 641 (K.A.)
Chodukin, N. J., 165 (K.A.)
—— & Sofieff, M. S., 155 (K.A.)
Chomereau-Lamotte, B., 50 (Misc.)
Chopra, R. N., 67 (Misc.), 279 (Dys.)
—— & Basu, U. P., 461 (Bb.)
—— & Choudhury, S. G., 61 (Misc.)
——, —— & Rao, S. S., 216 (Hel.)
—— & De, N., 267 (Am.)
—— & Knowles, R., 137 (Mal.)
Choremis, K., 92 (Fev.)
Chorine, V., with Marchoux, 495 *bis* (Z.), 738 (R.F.)
——, with —— & Markianos, 656 (Lep.)
Choudhury, S. G., with Chopra, 61 (Misc.)
——, with —— & Rao, 216 (Hel.)
Chowdhury, K. L., with Macdonald, 987 (Mal.)
——, with Roy, 112 (Mal.)
——, with Strickland, 943, 945 (Bl.)
Christopherson, J. B., 31, 75 (Misc.), 190 (Hel.)
Chtcherbakoff, S. G., 771 (Misc.)
Chu, F. T., Deitrick, S. & Chung, S. F., 732 (R.F.)
Chu, H. J., with Hegner, 482 *bis* (Z.)
Chukerbuti, J. C., 685 (Hel.)
Chung, H. & Kurotchkin, T. J., 642 (K.A.)
——, with ——, 399 (Der.)
—— & Reimann, H. A., 643 (K.A.)
Chung, S., with Chu & Deitrick, 732 (R.F.)
Ciaburri, G., (147) (Mal.)
Citron, H., 912 (S.S.)
Ciuca, M., Ballif, L. & Vieru, M., 119 (Mal.)
——, Irimesco, G., Ballif, L., Franke, M., Constantinesco, N. & Vieru, M., 136 (Mal.)
Clark, E. B., 203 (Hel.)
Clark, H. C., 483 (Z.)
—— & Dunn, L. H., 494 (Z.), 978 (Mal.)
Clarke, J. T., 34 (Misc.)
Clarke, T., 31 (Misc.)
Claude, H. & Coste, F., 630 (Fev.)
van Cleave, H. J., 780 (B.R.)
Cleland, J. B., 14, (866) (Misc.)
Clemesha, W. W. & Moore, J. H., 576 (Mal.)
Cleveland, L. R. & Collier, J., 486, 493 (Z.)
—— & Sanders, E. P., 791, 792 (Am.)
Clyde, D., 1025 (Mal.)
Cochrane, R. G., 649, 657 (Lep.)
Codounis, A., with Stefanopoulo, 295 (Y.F.)
Codvelle, Jausion & Dutrey, 349 (S.S.)
Cohen, A. J., with Rodenwaldt, 85 (Und.)
Cohen, J. B., with Browning, Ellingworth & Gulbransen, R., 925 (S.S.)
Colby, C. de W. & Schaffle, K., 232 (Hel.)
Collier, J., with Cleveland, 486, 493 (Z.)
Collier, W. A., 923 (S.S.)
—— & Warstadt, A., (1031) (Mal.)
——, with ——, 1013 (Mal.)
Colombo, 559 (Lab.)
Colomo de la Villa, G., with Suarez de Puga, 631 (Fev.)
Comaroff, R., with Geiger & Kligler, 374 (S.S.)
——, with Kligler & Geiger, 376 (S.S.)
Compton, A., 389, 394 (Pl.)
Conil, J., 9 (Bl.)
C[onnal], A., 48 (Misc.)
Connal, A., 554 (Lab.), 940 (Bl.)
——, with Glover, 8 (Bl.)
Connolly, P. P. D., 932 (Y. & S.)
Conseil, E., 101 (Fev.)
—— & Durand, P., (395) (Pl.)
——, with Durand, 98 (Fev.)
——, with Weiss & de Gentile, 966 (Oph.)
Constantinesco, N., with Ciuca, Irimesco, Ballif, Franke & Vieru, 136 (Mal.)
Conte, H. S. V., with Ferreira, 748 (Rab.)
Conterno, V., 702 (Und.)
Cook, A. B., with Low, (866) (Misc.)
Cook, C., 24 (Misc.)
Cooke, F. H., 650 (Lep.)
Cooley, T. B. & Lee, P., 40 (Misc.)
Copanaris, P., 322 (Lep.)
Corfield, C. E., with Self, (867) (Misc.)
Corke, W. H. & Bush, L. M., 843 (Misc.)
Cormack, R. P., with Jewell, 634 (Fev.)
Corman, 852 (Misc.)
Corpus, T., 773 (Misc.)
Corradini, G., (147) (Mal.)
Corson, J. F., 354, 369, 901, 902, 914 *bis*, 920 *bis*, 921 *bis* (S.S.)
Cort, E. C., 9 (Bl.)
Cort, W. W., with Schapiro, 174 (Hel.)
Costa Mandry, O. & Garrido Morales, E., 795 (Dys.)

da Costa, S. F. G., 177 *bis*, 178 *ter*, 181 (Hel.)
Costantini, H., (1031) (Mal.)
Coste, F., with Claude, 630 (Fev.)
Coutts, F. J. H., 829 (Hist.)
Couvy & Popoff, 839 (Misc.)
Couvy, L., 389 (Pl.)
—— & Popoff, 386 (Pl.)
Covell, G., 578, 973 (Mal.), 1035 (B.R.)
—— & Baily, J. D., 578 (Mal.)
Coventry, F. A., 491 (Z.)
Craig, C. F., 262 (Am.)
Craighead, A. C., 749 (Rab.)
Craste, 133 (Mal.)
Crawford, H. & Gutteridge, N. M., 662 (Sp.)
van Creveld, S., (692) (Hel.)
Croce, C., with Greenway, (552) (Z.)
Croll, D. G., 54 (Misc.)
Croste, R., 676 (Hel.)
Croveri, P., 22 (Misc.)
Crozat, P. & Assali, J., 838 (Misc.)
Cruz, M. C., with Samson & Lara, 329 (Lep.)
Cruz, O., Jr., with Godoy & Lobo, 586 (Mal.)
Cuboni, E. & Milani, C., (1031) (Mal.)
Cudlipp, J. S., 546 (Z.)
Cuff, C. H., 204 (Hel.)
Cumming, H. S., 883 (Chl.)
da Cunha, A. M., 646 (K.A.)
Curasson, G. & Dischamps, A., 742 (Rab.)
Curran, J. A. & Feng, S. T., 538 (Z.)
Cushing, E. C., with Laake, 528 (Z.)
Cusumano, A.. 262 (Am).

D

Dabbadie, P., 243 (Rab.)
Dagnino, V., (866) (Misc.)
Daleas & Haslé, 245 (Rab.)
Daleas, P., 544 (Z.)
Dalma, G. & Tuchtan, D., 605 (Mal.)
Dany, G., 381 (Pl.)
Dany, H., 874 (Pl.)
Das, K. K., (692) (Hel.)
Dassen, R. & Rey, J. C., (241) (Hel.)
Datta, S., 207 (Hel.)
Dau, H., with Pijper, 101, 636 (Fev.)
Daukes, S. H., 26 (Misc.)
David, N. A. & Leake, C. D., 852 (Misc.)
Davies, W. M., 527 (Z.)
Davis, G. E., 719 (Y.F.)
——, with Caldwell, F. C. & Caldwell, E. L., 222 (Hel.)
Davis, L. J., 685 (Hel.), 921 (S.S.)
Davis, N. C., 292, 717, 726 *bis*, 727 (Y.F.)
——, with Burke, 289 (Y.F.)
—— & Shannon, R. C., 727 (Y.F.)
Dawson, W. T., 816 (Hist.)
—— & Garbade, F. A., 62, 63 (Misc.)
Day, H. B., 31 (Misc.)
De, N., with Chopra, 267 (Am.)
De, S. C., 101 (Fev.)
Debono, J. E., with Zammit, 88 (Und.)
Dechaume, J., with Paviot & Martin, 87 (Und.)
Decourt, P., 999, (1031) (Mal.)
——, with Tanon & Cambessédès, 1032 (Mal.)
Deeks, W. E., 108, 120, 978 (Mal.)
Degos, R., with Milian, (658) (Lep.)
Deibel, H. K. & Elsbach, E. M., 930 (Y. & S.)
Deitrick, S., with Chu & Chung, 732 (R.F.)
De la Camara, P., 1011 (Mal.)
—— & Moraleda, C., 601, 1011 (Mal.)
De la Guardia, J., 420 (Myc.)
Delanoë, E., 654 (Lep.)
Delanoë, P., (241) (Hel.), 299, 300, 736 (R.F.), 449 (Y. & S.)
Del Favero, E., 104 (Fev.)
De Los Reyes, J. M., 456 (Bb.)
Denham, A. A., 203 (Hel.)
Deniel, with Sicé, Boisseau & Provost, 901 (S.S.)
Denney, O. E., 323, (658) (Lep.)
——, Hopkins, R. & Johansen, F. A., 327 (Lep.)
Deplanche, 971 (Oph.)
Deraniyagala, P. E. P., 540 (Z.)
Desai, V. M., with Wilkinson, Stubbs & Beharilal, 988 (Mal.)
Deschiens, R., 490 (Z.)
—— & Gourvil, E., 486 (Z.)
——, with Troisier, 209 (Hel.)
Des Essarts, J. Q., 535 (Z.)
Devata-Soupramanien, with Labernadie, 172 (Hel.)
Dévé, F., 676 (Hel.)
Dey, N. C., (450) (Y. & S.)
Dhar, D. R., 882 (Chl.)
——, Dhar, H. & Adhyee, P. C., 436 (Chl.)
Dhar, H., with Dhar, D. R. & Adhyee, 436 (Chl.)
Dhunjibhoy, J. E., 58 (Misc.)
Dhur-Roy, J. & Rakshit, A., (658) (Lep.)
Diamantopoulos, J., (622) (Fev.)
Dias, E., 379 (S.S.)
Dickens, P. F., (866) (Misc.)
Di Domizio, G., 369 (S.S.)
Dikshit, B. B. & Row, R. S. T. M., 959 (Lep.)
Diliberto, U., 1014 (Mal.)
Dilling, W. J., with Sadler & Gemmell, 848 (Misc.)
Di Lullo, O., 408 (Der.)
Dimitry, T. J., 654 (Lep.)
Di Natale, A., (611) (Mal.)
Dinger, J. E., 722 (Y.F.)
——, Schüffner, W. A. P. & Snijders, E. P., 291, 295 (Y.F.)
——, with —— & ——, 718 (Y.F.)
——, ——, —— & Swellengrebel, N. H., 290, 725 (Y.F.)
——, with Snijders, 728 (Y.F.)
—— & Verschaffelt, F., 314 (Lept.)
Dios, R. L. & Nopoff, R., 534 (Z.)
Dischamps, A., with Curasson, 742 (Rab.)
Dixey, M. B. D., (241) (Hel.), 650 (Lep.)
Dixon, D. S., with Low, 50 (Misc.)
Dobell, C., 705 (Z.)
Doerr, R. & Schmidt, G. W., 208 (Hel.)
Doggart, J. H. & Shapland, C. D., 969 (Oph.)
Dominici, A., 376 (S.S.)
—— & Rocca, G. C., 145 (Mal.)
Donatien, A., with Parrot & Lestoquard, 157 (K.A.)
van Doninck, A., 821 (Hist.)
Dormal, V. J., with Andrews & Johnson, 372 (S.S.)
Dorolle, 1000 (Mal.)
Dostrowsky, A., (648) (K.A.)
Doubrow, S. & Rousset, J., 234 *bis* (Hel.)

Douglass, M., with McKinley, 508 (Z.)
Dowling, G. B., with Ashford, 73 (Misc.)
——, with —— & McKinley, 425 (Myc.)
Drake-Brockman, R. E., (801) (Am.)
Drenowsky, A. K., 1009 (Mal.)
Du, S. D., 199 (Hel.)
Dubois, A., 735 (R.F.), 907, 922 (S.S.)
Dubois, C. & Sollier, N., 87, 704 (Und.)
Dufosse, 226 (Hel.)
Duggan, J. N. & Nanavati, B. P., 479 (Oph.)
Duguid, C., (658) (Lep.)
Duke, H. L., 346, 347, 896 (S.S.)
—— & Wallace, J. M., 365 (S.S.)
Dumont, P., with Van den Branden & Nélis, 305 (R.F.), 900 (S.S.)
Dunbar, L. & Stephens, E. D., 276 (Dys.)
Dunn, with Anderson, 381 (Pl.)
Dunn, L. H., with Clark, 494 (Z.), 978 (Mal.)
Dupas, J., 451 (C.Bu.)
Dupont, A., 160 (K.A.)
Dupuy, H., 243 (Rab.)
Durand, G., 210 (Hel.)
Durand, P., 629 (Fev.), 876 (Pl.)
—— & Conseil, E., 98 (Fev.)
——, with —— (395) (Pl.)
Duren, A. N., (296) (Y.F.)
Durieux, C., with Mathis, 303, 735 (R.F.)
Du Toit, J. A., (241) (Hel.)
Du Toit, P. J., 742 (Rab.)
Dutrey, with Codvelle & Jausion, 349 (S.S.)
Dwijkoff, P. P., 328 (Lep.)
Dychitan, T., 861 (Misc.)

E

Eales, N. B., 199 (Hel.)
Earle, W. C., 114 (Mal.)
Ebert, M. K., 4 (Bl.)
Edwards, F. W., 509 (Z.)
Eguchi, S. & Nishiyama, I., 203 (Hel.)
Egypt, 480, 972 (Oph.)
Ehara, I., with Minami, 215 (Hel.)
Ehrmann, R., (147) (Mal.)
Eichhorn, M., (692) (Hel.)
El-Dalgamony, A., with Ali, 962 (Lep.)
El Din, M. S., with Hassan, 181 (Hel.)
El-Kattan, M. A., 480, 967 (Oph.)
El Khani, 159 (K.A.)
Ellenbogen, V., 236 (Hel.)
Ellingworth, S., with Browning, Cohen & Gulbransen, R., 925 (S.S.)
Elliot, R. H., 478 (Oph.)
Elsbach, E. M., with Deibel, 930 (Y. & S.)
Emck, W. F., 822 (Hist.)
Emery, F. E. & Herrick, C. A., 224 (Hel.)
d'Engel, R., with Jancsò, 990, 998 (Mal.)
Epstein, D., 848 (Misc.)
Eskey, C. R., 871 (Pl.)
Esquier, G., (147) (Mal.)
Essed, W. F. R., (438) (Chl.)
Essex, H. E. & Bollman, J. L., (241) (Hel.)
——, Markowitz, J. & Mann, F. C., 542 (Z.)
Eubanas, F., 329 (Lep.)
Evans, A. M., (552) (Z.)
Eyermann, C. H. & Strauss, A. E., 133 (Mal.)
Eyre, J. W. H., 83 (B.R.)

F

Fabre, R., with Binet, 847 (Misc.)
Facio, L., 410 (Der.)
Fairley, K. D., 8 (Bl.)
Fairley, N. H., 679 (Hel.)
——, with Low, 290 (Y.F.)
——, Mackie, F. P. & Jasudasan, F., 196(Hel.)
Falcão, E. de C., (241) (Hel.), 432 (Chl.), 477 (Oph.)
Far Eastern Association of Tropical Medicine, 169 (Hel.)
Farmakidis, C., (280) (Dys.), 787 (Am.), (878) (Pl.)
Fast, G., 930 (Y. & S.)
Faust, E. C., 198, 689, 690 (Hel.), 268 (Am.), 538 (Z.), 784 (Am.)
——, with Kagy, 793 (Am.)
—— & Kellogg, C. R., 171 (Hel.)
Fawcett, H. A., 531 (Z.)
Federated Malay States, 111, 985 (Mal.), 561 (Lab.)
Feegrade, E. S., 82 *bis* (Misc.)
Feemster, R., with Silverman, 797 (Dys.)
Feldmann, I., 208 (Hel.)
Feliciano, R. T., with Vedder, (464) (Bb.)
Feng, L. C., 667 (Hel.)
Feng, S. T., with Curran, 538 (Z.)
Ferradas, M. G., 1013 (Mal.)
Ferreira, A. A. & Conte, H. S. V., 748 (Rab.)
Ferrell, J. A., 991 (Mal.)
de Figueiredo, J. M. P., 382 (Pl.)
Findlay, G. M., 31 (Misc.)
—— & Hindle, E., 293 (Y.F.)
Finkelstein, M. H., 884 (Chl.)
Fischer, I. A., 309 (Lept.)
Fischer, O., 349 (S.S.)
Fischer, W. O., 405 (Der.)
Fishback, H. R., 225 (Hel.)
Fittipaldi, C., 490 (Z.)
Flatow, E., 823 (Hist.)
Fleming, N., 969 (Oph.)
Fletcher, W., 94 (Fev.)
Foley, H., with Joyeux, 202 (Hel.)
——, with Sergent, Edm., Sergent, Et., Parrot, Catanei & Senevet, 984 (Mal.)
Fonquernie, J., 383, 875 (Pl.)
da Fonseca, F., with Pinto, 527 (Z.)
da Fonseca, O., 400 (Der.)
—— & Levy, A. S., 399 (Der.)
da Fonseca, O., Jr., (415 *bis*) (Der.), (1031) (Mal.)
—— & Leão, A. E. de A., 404 (Der.)
—— & da Rosa, A. F., 401 (Der.)
Fontoynont, M., Girard, G. & Woltz, H., 935 (Y. & S.)
Forbes-Brown, A., 839 (Misc.)
Forbes, J., 12 (Bl.)
Fornara, with Arnaud, Vigoni & van Hoof, 932 (Y. & S.)
Forsyth, W. L. & Gohar, M. A., 310 (Lept.)
Fort, M. A., 774 (Misc.)
Fotheringham, W. T., with Ruiz, 452 (C.Bu.)
Found, N., 19 (Misc.)
Fourneau, E., Tréfouel, M. & Mme. & Benoit, G., 64 (Misc.)
——, ——, Stefanopoulo, G., Benoit, G., de Lestrange, Y. & Melville, K. I., 64 (Misc.)
Fowler, J. K., 286 (Y.F.)

Fox, H., 396 (Der.), 648 (K.A.)
Foy, H. A., 887 (H.S.)
Fradkin, W. Z. & Schwartz, L. S., 41 (Misc.)
Fraga, A., 398 (Der.)
Fraga, C., et al, 714 (Y.F.)
France: Ministère de la Guerre, 869 (B.R.)
Franchini, G., 116, (147) (Mal.), 298 (R.F.)
—— & Pirami, E., 643 (K.A.)
—— & Taddia, L., 732 (R.F.)
Francisco, S. A., with Tubangui, 199 (Hel.)
Franco, E. E., 58 (Misc.)
—— & Manai, A., 149 (K.A.)
Frank, A. W., (147) (Mal.)
Franke, M., with Ciuca, Irimesco, Ballif, Constantinesco & Vieru, 136 (Mal.)
Frazier, C. N., Kurotchkin, T. J. & Mu, J., 418 (Myc.)
Freed, H., with Gallagher, (866) (Misc.)
Freeman, H., with Ingall, 203 (Hel.)
Frégonneau, W., 113 (Mal.)
Frei, W. & Wiese, J., 937 (C.Bu.)
de Freitas, O., 256 (Rab.)
Freudenthal, W., 452 (C.Bu.)
Freyd, A., 23 (Misc.)
Frobisher, M., Jr., 720 *bis*, 721 (Y.F.)
Fróes, H. P., 694 (B.R.)
Fülleborn, F., 235, 236 (Hel.)
—— & Kikuth, W., 234 (Hel.)
Fullerton, H. R., 1027 (Mal.)
Fulmer, E. I., with Buchanan, 168 (B.R.)
Fuse, S., 409 (Der.)

G

Gabbi, U. & Mariotti-Bianchi, 26 (Misc.)
Gadaud, with Louste & Lévy-Franckel, (658) (Lep.)
Gaisky, N., 393, 877 (Pl.)
Gaitan, L., 282 (Y.F.)
Gallagher, E. T. & Freed, H., (866) (Misc.)
Gallea, M., 249 (Rab.)
Gambia, 543 (Z.)
Gambier, 195 (Hel.)
Gambier, A., 39 (Misc.), 172 (Hel.), 264 (Am.) (415) (Der.)
Ganora, R., 24, 69 (Misc.), (147) (Mal.)
Garbade, F. A., with Dawson, 62, 63 (Misc.)
Garcia Maldonado, L., 452 (C.Bu.)
Garcia de Obeso, L., 769 (Misc.)
Garcia, O., with Schöbl, 443 (Y. & S.)
Gardner, G., 1035 (B.R.)
Gardner, L., with Starr, 663 (Sp.)
Garin, C., Rousset, J. & Gonthier, B., 233 (Hel.)
Garnham, P. C. C., 995, 1020 (Mal.)
Garnier, M., Nicaud, P. & Maisler, A., (740) (Lept.)
Garrido Morales, E., with Costa Mandry, 795 (Dys.)
Garry, G., 407 (Der.)
Gasca, A., 764 (Misc.)
Gasperini, C. G., 162, 645 (K.A.)
Gater, B. A. R., 610 (Mal.), 834 (Misc.)
Gatowskaja, R. G. & Kasakoff, P. T., 208 (Hel.)
Gautier, R., 437, 879 (Chl.)
Gay, D. M. & Bigelow, J. B., 402 (Der.)
Gebeili, E., (878) (Pl.)
Geiger, A., Kligler, I. J. & Comaroff, R., 374 (S.S.)
——, with —— & ——, 376 (S.S.)
Geiger, J. C. & Gray, J. P., (1031) (Mal.)
Gelli, G., with Giordano, 182 (Hel.)
Gemmell, A. A., with Sadler & Dilling, 848 (Misc.)
Geneeskundig Tijdschrift voor Nederlandsch-Indië, 563 *bis*, 564 (Lab.)
de Gentile, O. R., with Weiss & Conseil, 966 (Oph.)
George, I. D., 546 (Z.)
Georgi, F. & Beyer, A., 467 (Pel.)
Gerbasi, M., 786, (801) (Am.)
Gerber, C. W., 991 (Mal.)
Gessner, O., (280) (Dys.)
Gesteira, M., 130 (Mal.)
Gharpuré, P. V., 156 (K.A.)
—— & Saldanha, J. L., 783 (Am.)
Gheorghiu, I., 279 (Dys.)
Ghosh, B. N., 779 (B.R.)
Giemsa, 60 (Misc.), 357 (S.S.)
—— & Jírovec, O., 912 (S.S.)
Giglioli, G., 42 (Misc.), 131 (Mal.), 454 (G.V.)
Gilder, W., with Makel, 12 (Bl.)
Gilks, 134 (Mal.)
Gill, C. A., 106, 996 (Mal.)
—— & Lal, R. B., 879 (Chl.)
Gillispie, A. M., 951 (Bl.)
Gilmour, C. C. B., 856 (Misc.)
Gingold, N., with Nicolau, 998 (Mal.)
Giordano, M. & Gelli, G., 182 (Hel.)
——, with Cesarano, (552) (Z.)
Girard, G., 386, 878 (Pl.)
——, with Fontoynont & Woltz, 935 (Y. & S.)
—— & Hérivaux, A., 653 (Lep.)
Giraud-Costa, E., 627 (Fev.)
Girges, R., 195 *bis*, (241) (Hel.)
Girolamo, L., 31 (Misc.)
Glaubersohn, S. A. & Goldenberg, M. M., 471 (Pel.)
Glover, W. E. & Connal, A., 8 (Bl.)
Glusman, M. P. & Goldenberg, J. I., 256 (Rab.)
——, Sadowsky, I. J. & Solowjewa, J. W., 244 (Rab.)
—— & Solowjewa, J. W., 746 (Rab.)
——, Solowjewa, J. W. & Predtetschenskaja, L. A., 244 (Rab.)
Godoy, A., Lobo, A. & Cruz, O., Jr., 586 (Mal.)
Gohar, M. A., 960 (Lep.)
——, with Forsyth, 310 (Lept.)
Goiran, E., with Blanc, (1031) (Mal.)
Goldberger, J. & Sebrell, W. H., 473 (Pel.)
——, Wheeler, G. A., Rogers, L. M. & Sebrell, W. H., 473 (Pel.)
Golden, R., with O'Connor & Auchincloss, (692) (Hel.)
Goldenberg, I., with Zuwerkalow, 246 (Rab.)
Goldenberg, J. I., with Glusman, 256 (Rab.)
Goldenberg, M. M., with Glaubersohn, 471 (Pel.)
Goldina, R. B., with Gubin, 247 (Rab.)
Golov, D. & Kniazevsky, A., 393 (Pl.)
Golovine, S., 673 (Hel.)
Golow, D. & Knjasewskii, A., 383 (Pl.)
Golubewa, E. E., with Rubinstein, 70 (Misc.)
Gomes, J. M., 961 (Lep.)
Gomez, L. B., 331 (Lep.)

Gomila, F. R., 388 (Pl.)
Gonthier, B., with Garin & Rousset, 233 (Hel.)
Gonzalez, H. E., Ontaneda, L. E. & Vidaurreta, M., 162 (K.A.)
Goodale, R. H., 676 (Hel.)
—— & Krischner, H., 210 (Hel.)
Goodner, K. & Shattuck, G. C., 773 (Misc.)
——, with Shattuck, 447 (Y. & S.)
Goodson, J. A. & Henry, T. A., 61 *bis* (Misc.)
——, —— & Macfie, J. W. S., 849 (Misc.)
Goodwin, T. S., 184 (Hel.)
Gool, A. H., 768 (Misc.)
Gopsill, W. L., 173, 188 (Hel.)
Gordon, R. M., 70 (Misc.)
—— & Hicks, E. P., 192 (Hel.)
Gosio, R., 1031 (Mal.)
Götze, G., with Schander, 835 *bis* (Misc.)
Goubarev, E. & Lipatova, T., 877 (Pl.)
Gougerot & Aubin, 324 (Lep.)
Gourevitsch, D., 490 (Z.)
Gourmelon, (13) (Bl.)
Gourvil, 786 (Am.)
Gourvil, E., with Deschiens, 486 (Z.)
Gover, W., 662 (Sp.)
Goyle, A. N., with Taylor, 739 (Lept.)
Grabow, C., with Plaut, 301 (R.F.)
Gracias, J. B. A., 810 (Hist.)
Gradwohl, R. B. H., 71 (Misc.)
Graham, G. M., with Weller, 299 (R.F.)
Graham, J. D., 381 *bis* (Pl.), 431, 883 (Chl.)
Graham, J. W., 38 (Misc.)
Graham-Smith, G. S., 527 (Z.)
Grant, F., 200 (Hel.)
Grant, P. F. A., with Thompson, 121 (Mal.)
Gratch, I., 115 (Mal.)
Graterol y Morles, J., (464) (Bb.)
Gray, G. M., 48 (Misc.)
Gray, J. P., with Geiger, (1031) (Mal.)
Green, R., 143 (Mal.)
Green-Armytage, V. B., 831 (Hist.)
Greenway, D. & Croce, C., (552) (Z.)
Greer, A. E., 469 (Pel.)
Greig, D. M., 545 (Z.), 753 (Rab.)
Greval, S. D. S., with Taylor & Thant, 273 (Dys.)
Grey, J. C. P., 231 (Hel.)
Griffiths-Jones, E., Atkinson, H. & Hassan, A., 668 (Hel.)
Griffitts, T. H. D., 108 (Mal.)
Grigorowa, O. & Nesturch, M., 218 (Hel.)
Grinker, R. R., 749 (Rab.)
——, with Bassoe, 245 (Rab.)
Gritzai, P. K., 513 (Z.)
Gron, K., 334 (B. R.)
Gros, H., 830 (Hist.)
Grünbaum, F. T., with Kritschewski & Schapiro, 854 (Misc.)
Gualdi, A., 150 (K. A.)
Guardia, G. T., 52 (Misc.)
Gubin, W. M. & Goldina, R. B., 247 (Rab.)
Gueliadow, N. B., 138 (Mal.)
Guerricchio, A., 165 (K.A.)
Gulbransen, R., with Browning, Cohen & Ellingworth, 925 (S.S.)
Gulino, M., 626 (Fev.)
Gupta, B. M. D., 154, 163, 646 (K.A.), 595 (Mal.)
——, with Knowles, 129, 588 (Mal.)
Gupta, C. R. D., with Napier, 160 (K.A.)
Gupta, D., with Knowles & White, (611) (Mal.)
Guthrie, R. H., 470 (Pel.)
Gutteridge, N. M., with Crawford, 662 (Sp.)
Guy, R., (1032) (Mal.)

H

Haberfeld, W., (866) (Misc.)
Hackett, L. W., 124, 976 (Mal.)
—— & Missiroli, A., 569 (Mal.)
van Haeften, J., with Hamilton & Basu, 886 (H.S.)
Haga, J., 217 (Hel.)
——, with Brug, 536 (Z.)
Hakim, A., 993 (Mal.)
Hakki, I., 176 (Hel.)
Hakushi, R., with Koidzumi, (552) (Z.)
Halawani, A., 268 (Am.)
Haldar, K. C., with Napier, 161 (K.A.)
Hall, M. C., 177 *bis* (Hel.)
Haller, O., 394 (Pl.)
Hamerton, A. E., 892 (S.S.)
Hamilton, C. S. P., Basu, D. N. & van Haeften, J., 886 (H.S.)
Hamlyn-Harris, R., 503 *bis* (Z.)
Hancock, G. L. R., 502 (Z.)
Handbuch der Haut- und Geschlechtskrankheiten, 334 (B.R.)
Hanson, H., 109 (Mal.)
Hanzawa, S., 49 (Misc.)
Hara, Y., 552 (Z.)
Harbhagwan, with Sinton & Singh, 587 (Mal.)
Hare, H. A., 819 (Hist.)
Hare, T., 226 (Hel.)
Haridas, G., 44, 841 (Misc.)
Harman, N. B., 475 (Oph.)
Harris, R. H. T. P., 524, 525 (Z.)
Harris, R. M., (664) (Sp.)
Harston, M., 662 (Sp.)
Hartmann, E., 373 (S.S.)
Hartsock, F. M., (866) (Misc.)
Harvard Institute for Tropical Biology and Medicine, 258 (B.R.)
Harvey, W. F., 27 (Misc.)
Hase, A., 528 (Z.)
Hasegawa, K., 206 *bis* (Hel.)
Haslé, with Daleas, 245 (Rab.)
Haslé, G., (147) (Mal.)
—— & Nguyên-Duy-Ha, 138 (Mal.)
——, with Normet & Nguyên-Duy-Ha, 407 (Der.)
Hassan, A. & El Din, M. S., 181 (Hel.)
——, with Griffiths-Jones & Atkinson, 668 (Hel.)
Hasselmann, C. M., 447 (Y. & S.), 885 (Chl.)
—— & Miyao, I., 413 (Der.)
Hasselmann-Kahlert, M., 956 (Lep.)
Hässler, E., 798 (Dys.)
Havens, L. C. & Castles, R., 229 (Hel.)
Hawaii, 650 (Lep.)
Hawe, J. A. & Russell, H., 244 (Rab.)
Hawking, F., with Yorke & Murgatroyd, 909, 910 (S.S.)
Hayasaka, E. & Inawashiro, R., 459 (Bb.)
Heckenroth, F. & Advier, M., 674 (Hel.)
v. Hecker, H. & Kellner, F., 205 (Hel.)
Hegh, E., 526 (Z.)

Hegner, R. & Chu, H. J., 482 *bis* (Z.)
Heinemann, H., 589 (Mal.), 836 (Misc.)
——, Koumans, A. K. J. & Pirngadi, R., 30 (Misc.)
Hellerström, S., 937 (C.Bu.)
—— & Wassèn, E., 938 (C.Bu.)
Hellinga, G., 822 *ter* (Hist.)
Helmy, M. M., (242), 669 (Hel.)
Hemenway, R. V., (611) (Mal.)
Henderson, J. M., 963 (Lep.)
—— & Kelly, F. C., 69 *bis* (Misc.)
——, with ——, 69 (Misc.)
Henry, A. F. X., 139, (611), 1023 (Mal.)
——, with Le Bourdelles, 140 (Mal.)
Henry, T. A., with Goodson, 61 *bis* (Misc.)
——, with —— & Macfie, 849 (Misc.)
d'Herelle, F., 879 (Chl.)
Herivaux, 450 (Y. & S.)
Hérivaux, A., 411 (Der.), 962 (Lep.)
——, with Girard, 653 (Lep.)
Hermans, E. H., 414 (Der.)
Hermant, 434 (Chl.)
Hernandez, L. G., 664 (Sp.)
Hernandez, V., with Butler, 828 (Hist.)
Herrero Rubio, P., 164 (K. A.)
Herrick, C. A., with Emery, 224 (Hel.)
Herrmann, O., 255, 748 (Rab.)
Herzberg, K., with Manteufel, 729 (Y.F.)
Hesse, E., 749 (Rab.)
Heubner, D. L., 853 (Misc.)
van Heurn, W. C., 79 (Misc.)
Heyd, C. G. & Sheplar, A. E., (280) (Dys.)
Hicks, E. P., 532 (Z.)
——, with Gordon, 192 (Hel.)
Higoumenakis, G., 167 (B.R.)
Higuchi, S., 311 (Lept.)
Hindle, E., 285 (Y.F.)
——, with Findlay, 293 (Y.F.)
Hinman, E. H., 506 (Z.)
Hinrichsen, H. M., (242) (Hel.)
Hirose, Y., 321 (Lep.)
Hirst, L. F., 559 (Lab.)
Hissette, J., 970 *bis* (Oph.)
Hiyeda, K., 269 (Am.)
Hoang-Su, 135 (Mal.)
Hoeppli, R. J. C., (242) (Hel.)
Hoffman, W. A., 674 (Hel.)
——, with Taliaferro, 214 (Hel.)
Hoffmann, C. C., 219, 684 (Hel.)
Hoffmann, W. H., (296 *bis*), (729) (Y.F.)
Hoffstadt, R. E. & Lingenfelter, J. S., 421 (Myc.)
——, with Wheelon, 422 (Myc.)
Hofkamp, H. S., with Nieschulz, 222 (Hel.)
Holder, H. G., Osborne, C. J. & Sommermeyer, V., 791 (Am.)
Honna, F., 788 (Am.)
van Hoof, with Arnaud, Fornara & Vigoni, 932 (Y. & S.)
d'Hooghe, M., 128 (Mal.)
Hopkins, H. O., (1032) (Mal.)
Hopkins, R., with Denney & Johansen, 327 (Lep.)
Horn, A. E., (866) (Misc.)
Horn, L. & Kauders, O., 57 (Misc.)
Hornby, H. E. & Bailey, H. W., 924 (S.S.)
Hoshi, N., (280) (Dys.)
Houssiau, 266 (Am.)
Houssiau, F., with Rodhain, 219 (Hel.)
Howland, L. J., 505 (Z.)
Hoyt, A. & Jungeblut, C. W., 752 (Rab.)
Hsieh, C. K., with Keefer, 460 (Bb.)
Hu, C. K., with Yang, 466 (Pel.)
Huang, K. K., with Keefer & Yang, 797 (Dys.)
Huard, P., with Botreau-Roussel, 685 (Hel.)
Huart, A. J., 233 (Hel.)
Hubert, J., with Hübin, 968 (Oph.)
Hübin, R. & Hubert, J., 968 (Oph.)
Huddleson, I. F., 700 (Und.)
Hudson, N. P., 858 (Misc.)
—— & Kitchen, S. F., 284 (Y.F.)
Huff, C. G., 507 (Z.)
Hughes, T. A., 1006 (Mal.)
—— & Shrivastava, D. L., 598 (Mal.)
Huie, D., with Yao & Yuan, 43 (Misc.)
Hume, E. E., 408 (Der.)
Hurst, A. F., with Armand-Delille & Sorapure, 56 (Misc.)
Hussameddin, 616 (Fev.)
Hutchison, W., with Le Sueur, 409 (Der.)
van Huyen, P., 255 (Rab.)
——, with Verge, 255 (Rab.)
Hval, E., (453) (C.Bu.)
Hynd, D., 768 (Misc.)

I

Ibrahim, A., 412 (Der.)
Ichok, G., 126 (Mal.)
Ida, T., with Takahashi, Ishikawa & Ogawa, 467 (Pel.)
Imms, A. D., 259 (B.R.)
Imperial Institute of Entomology, 499 (Z.)
In, K., 397, (Der.), 417 (Myc.)
Inawashiro, R. & Hayasaka, E., 459 (Bb.)
India, 15 (Misc.)
Indian Medical Gazette, 611 (Mal.)
Ingall, M. & Freeman, H., 203 (Hel.)
Innes, F., (801) (Am.)
Innes, F. A., 543 (Z.)
Inoue, S., 313 (Lept.)
Ionesco, D., 750 (Rab.)
Irimesco, G., with Ciuca, Ballif, Franke, Constantinesco & Vieru, 136 (Mal.)
Isaacs, M., with Caius & Mhaskar, 68 (Misc.)
Ishikawa, K., with Takahashi, Ogawa & Ida, 467 (Pel.)
Ishikawa, S., 225 (Hel.)
Ishiwara, K. & Ogata, N., 104 (Fev.)
Isotti, F., 453 (C.Bu.), 537 (Z.)
Itabashi, K., (753) (Rab.)
Iwanowsky, N. & Sassykina, T., 391 *bis* (Pl.)
Iyengar, with Asheshov, Taylor, Morison & Malone, 863 (Misc.)
Iyengar, K. R. K. & Beer, W. A., 249 (Rab.)
Iyengar, M. O. T., 111, 980 (Mal.)
——, with Rao, 212 (Hel.)
Iyer, P. V. S., with King, Pandit & Menon, 211 (Hel.)
——, with Pandit, C. G. & Pandit, S. R., (242) (Hel.)
Izar, G., 149 (K.A.)
Izumita, T., with Ohta, 459 (Bb.)

J

Jack, R. W., 522, 523 *bis* (Z.)
Jackson, C., with Thomas, 744 (Rab.)

Jackson, C. H. N., 521 (Z.)
Jackson, R. B., 129 (Mal.)
Jacobi, A., with Jutz, 605 (Mal.)
Jadin, J., with Vassiliadis, 306 (R.F.), 370 (S.S.)
Jaensch, P. A., 469 (Pel.)
Jamal-ud-Din, (103) (Fev.)
James, J. F., 1030 (Mal.)
James, S. P., 287 *bis* (Y.F.), 566 (Mal.)
James, W. M., 52 (Misc.)
James, S. P., Nicol, W. D. & Shute, P. G., 973 (Mal.)
Jancsó, N. & d'Engel, R., 990, 998 (Mal.)
Jankelson, I. R., 1000 (Mal.)
Jankoschwili, W. & Karibow, N., 176 (Hel.)
Jared, D., with Marshall, 89 (Und.)
Jasudasan, F., with Fairley & Mackie, 196 (Hel.)
Jausion, with Codvelle & Dutrey, 349 (S.S.)
Jausion, H. & Sohier, R., 416 (Myc.)
Javett, S. N., 600 (Mal.)
Jeanselme, 324 (Lep.), 825 (Hist.)
Jemma, R., 641 (K.A.)
Jewell, N. P. & Cormack, R. P., 634 (Fev.)
Jewett, C. H., with Baumgartner, 659 (Sp.)
Jírovec, O., with Giemsa, 912 (S.S.)
Joannidès, G., with Blanc & Papaïoannou, 404 (Der.)
Joannidès, G. S., 618 (Fev.)
Joannidès, N. Z., 648 (K.A.)
Joenoes, M., with Parjona & Radsma, 862 (Misc.)
Johannessohn, F., 167 (B.R.)
Johansen, F. A., with Denney & Hopkins, 327 (Lep.)
Johns, F. M., 1024 (Mal.)
Johnson, C. M., with Andrews & Dormal, 372 (S.S.)
Johnson, H. A., with Leprince, 80 (Misc.)
Johnson, W. B., 48, 754 (Misc.), 341 (S.S.)
Johnston, H. W., with Power, 226 (Hel.)
Johnston-Saint, P., 808 (Hist.)
Jolly, 381 (Pl.)
Jolly, G. G., 456 (Bb.)
Joltrain, 874 (Pl.)
Jones, J. W. & Alden, H. S., 402, 404 (Der.)
Jones, W. J. & Bunting, C. H., (692) (Hel.)
Jonesco, D., 255 *bis* (Rab.)
Jordan, E. O., 1036 (B.R.)
Jorge, R., 98 (Fev.), 281, (296) (Y.F.), 997 (Mal.)
Journal de Médecine de Bordeaux, 830 (Hist.)
Journal of the Medical Association of South Africa, 768 *bis* (Misc.)
Jowelew, B. M., 746 (Rab.)
Joyeux, B., with Bablet & Leclerc, 245 (Rab.)
Joyeux, C. & Foley, H., 202 (Hel.)
—— & Pieri, J., 630 (Fev.)
Jungeblut, C. W., with Hoyt, 752 (Rab.)
Junior, J. S., with Shannon, (553) (Z.)
Jutz, B. & Jacobi, A., 605 (Mal.)

K

Kagaya, J., with Tanaka, Kaiwa & Teramura, 103 (Fev.)
Kagy, E. S. & Faust, E. C., 793 (Am.)
Kaiser, L., 443 (Y. & S.)
Kaiser, M., with de Lavergne & Robert-Lévy, 311 (Lept.)
Kaiwa, J., with Tanaka, Teramura & Kagaya, 103 (Fev.)
Kakishita, M., 447 (Y. & S.)
Kalajev, A. W., with Brussin, 919 (S.S.)
Kalandadse, W. & Mtschedlidse, I., 539 (Z.)
Kalandadze, L., 510 (Z.)
Kalina, G. P., (878) (Pl.)
Kamisaka, T., 673 (Hel.)
Kamizawa, O., with Chin-Kyu-Sui & Shimokawa, (866), (867) (Misc.)
Kanagarayer, K., 26 (Misc.)
Karibow, N., with Jankoschwili, 176 (Hel.)
Karłowski, Z., 251 (Rab.)
Kasakoff, P. T., with Gatowskaja, 208 (Hel.)
Kasauli, 749 (Rab.)
Katagai, T., 533 (Z.)
Katz, D. I., 298 (R.F.)
Kauders, O., with Horn, 57 (Misc.)
Kawamura, H., 734 (R.F.)
Kazariane, A. D., 12 (Bl.)
Keatinge, L. R. H., (866) (Misc.)
Keefer, C. S. & Hsieh, C. K., 460 (Bb.)
——, Yang, C. S. & Huang, K. K., 797 (Dys.)
Kehar, N. D., with Sinton, 508 (Z.), 1024 (Mal.)
Kellaway, C. H., 546, 547, 548 (Z.)
—— & Thomson, D. F., 547 (Z.)
Kellner, F., with v. Hecker, 205 (Hel.)
Kellogg, C. R., with Faust, 171 (Hel.)
Kelly, F. C. & Henderson, J. M., 69 (Misc.)
——, with——, 69 *bis* (Misc.)
Kelser, R. A., 747 (Rab.)
Kemper, H., 501 (Z.)
Kenaoui, N., 394 (Pl.)
Kendrick J. F., 169 (Hel.)
Kenya & East African Medical Journal, 134 (Mal.), 689 (Hel.)
Kerbler, F., 747 (Rab.)
Kerbosch, M., 817 (Hist.)
Kerr, J. A., 722 (Y.F.)
Kestner, O. & Borchardt, W., 862 (Misc.)
Keukenschrijver, N. C. R., 237 (Hel.)
Khalil, M., 184 *bis*, 665 (Hel.), 540 (Z.), 698, 952 (B.R.)
Khan, S., 436 (Chl.)
——, with Asheshov & Lahiri, 880 (Chl.)
Khaw, O. K., 184 (Hel.), 641 (K.A.)
Khouri, J., (242) (Hel.)
Kidd, F., 216 (Hel.)
Kikuth, W., with Fülleborn, 234 (Hel.)
Kilbourne, E. D., 266 (Am.)
Kimsan with Quenardel, (801) (Am.)
King, with Russell & Pandit, 381 (Pl.)
King, H. H., Pandit, C. G., Menon, K. P. & Iyer, P. V. S., 211 (Hel.)
King, W. V., 109 (Mal.)
Kingsbury, A. N., 561 (Lab.)
Kirby, 477 (Oph.)
Kirby, D. B. & Newell, W. G., 969 (Oph.)
Kirk, J. B., 260, 336 (B.R.)
Kirk, N. T., (964) (Lep.)
Kirschner, L., 36 (Misc.)
Kister, 384 (Pl.)
Kitamura, K., 236 (Hel.)
Kitchen, S. F., with Hudson, 284 (Y.F.)
Kleine, F. K., 30, 765 (Misc.), 893 (S.S.)
Kleine, H. O., 824 (Hist.)

Kligler, I. J., 602, (1032) (Mal.)
——, Geiger, A. & Comaroff, R., 376 (S.S.)
——, with—— & ——, 374 (S.S.)
—— & Mer, G., 589, 599, 1008 (Mal.)
Klingmuller, V., 334 (B.R.)
Klotz, O., 217 (Hel.)
—— & Belt, T. H., 287, 288 *ter* (Y.F.)
Klövekorn, G. H., 322 (Lep.)
Knabe, K., 866 (Misc.)
——, with Mühlens, 947 (Bl.)
Kniazevsky, A., with Golov, 393 (Pl.)
Knights, E. M., 146 (Mal.)
Knjasewskii, A., with Golow, 383 (Pl.)
Knott, J., 928 (Y. & S.)
Knowles, R., 1019 (Mal.)
——, with Chopra, 137 (Mal.)
—— & Gupta, B. M. D., 129, 588 (Mal.)
—— & White, R. S., 612 (B.R.)
——, —— & Gupta, D., (611) (Mal.)
Knutti, R. E., with Olitsky & Tyler, 966 (Oph.)
Kobayashi, H., 202, 677 *ter*, 678, (692) (Hel.)
Koch, D. A., with Anderson, 791 (Am.)
Koch, E., with Aalsmeer, 460 (Bb.)
Koidzumi, M. & Hakushi, R., (552) (Z.)
Kolmer, J. A., 361 (S.S.)
——, with Rule, A. M., 907 (S.S.)
Komendantov, V., (147) (Mal.)
Kopciowska, L., with Nicolau, 247 *bis* (Rab.)
——, with—— & Viala, 246 (Rab.)
Kopeloff, N., 142 (Mal.)
Kopke, A., 345 (S.S.)
Kopstein, F., 545 (Z.)
Korke, V. T., 170, 211 *ter* (Hel.)
Korteweg, P. C., 591, 992 (Mal.)
Korthof, G., 309 (Lept.)
——, with Bijl, 308 (Lept.)
Kotake, M., (242) (Hel.)
Koumans, A. K. J., with Hememann & Pirngadi, 30 (Misc.)
Kouwenaar, W., 42, 772 (Misc.)
Kreidel, K. V., with Wollstein, 40 (Misc.)
Kremer, B., 622 (Fev.)
Krikorian, K. S., with Stuart, 253 (Rab.)
Krischner, H., with Goodale, 210 (Hel.)
Kritschewski, I. L., 304 (R.F.)
——, Grünbaum, F. T. & Schapiro, S. L., 854 (Misc.)
——, Semzova, O. M. & Ratner, M. I., 855 (Misc.)
—— & Sinjuschina, M. N., 737 (R.F.)
Krumberg, A. J., with Rudnew, 703 (Und.)
Kubokawa, K., with Tôyama & Miyoshi, 332 (Lep.)
Kudo, R. R., 693 (B.R.)
Kudrjawzew, W. & Scensnowic, W., 666 (Hel.)
von Kühlewein, M., 17 (Misc.)
Kumm, H. W., 858 (Misc.)
——, with Beeuwkes & Walcott, 858 (Misc.)
Kurauchi, K., 387 *bis* (Pl.)
——, with Ando & Nishimura, 383 (Pl.)
Kuriyan, A. T., 686 (Hel.)
Kurotchkin, T. J. & Chen, F. K., 396 (Der.)
—— & Chung, H. L., 399 (Der.)
——, with ——, 642 (K.A.)
——, with Frazier & Mu, 418 (Myc.)
—— & Lim, C. E., 421 (Myc.)
——, with ——, 420 (Myc.)
Kusnetzow, W. N., (658) (Lep.)
Kuyer, A., 633 (Fev.)
Kyriasides, K. N., 403 (Der.)
Kyriasidis, K. N., 118 (Mal.)
Kyriazidès, K., 403 (Der.)

L

Laake, E. W. & Cushing, E. C., 528 (Z.)
Labbé, M., 265 (Am.)
Labernadie, V., 824 (Hist.)
—— & Devata-Soupramanien, 172 (Hel.)
—— & Narayanin, C., (438) (Chl.)
Labernadie, V. G. F., 169 (Hel.)
Lacapère, G., 933 (Y. & S.)
Ladyjenski, P. M., 588 (Mal.)
Lages, J. K. da C., (147) (Mal.)
Lahiri, M. N., with Asheshov & Khan, 880 (Chl.)
Laignel-Lavastine, M., Boquien, Y. & Puymartin, (740) (Lept.)
Laing, F., 518 (B.R.)
Lal, R. B., with Gill, 879 (Chl.)
Lamakine, 253 (Rab.)
Lambert, J. M., 14 (Misc.)
Lambert, S. M., 928 (Y. & S.)
Lamborn, A. W., 498 (Z.)
Lamy, P., 282 (Y.F.)
Lan-Chou, F., 218 (Hel.)
Landeiro, F., 961 (Lep.)
Lane, C., 66 (Misc.), 227, 228 (Hel.), 979 (Mal.)
de Langen, C. D., 207 (Hel.)
Langeron, M., 416 (Myc.)
—— & du Noyer, M. R., 696 (B.R.)
Laquièze, 956 (Lep.)
Lara, C. B., 328 (Lep.)
——, with Samson & Cruz, 329 (Lep.)
La Rosa, G., 390 (Pl.)
Larrousse, F., with Borrel, 658 (Lep.)
Lasnet, 297 *bis* (R.F.)
Latko, N. & Michailow, S., 938 (C.Bu.)
Launoy, L. & Prieur, M., 362 *bis*, 906, (S.S.)
Laurel, A. G., 510 (Z.)
Laurinsich, A., 643 (K.A.)
Lauterburg, M., 353 (S.S.)
——, with Stalder, 867 (Misc.)
Lavergne, J. & Monier, H., 1022 (Mal.)
de Lavergne, V., Robert-Lévy & Kaiser, M., 311 (Lept.)
Lavier, G., 847 (Misc.)
Lazaro Caballero, R. & Pastor Botija, F., 164 (K.A.)
League of Nations, 345 (S.S.), 571 (Mal.), 953 *bis* (Lep.)
League of Nations Monthly Epidemiological Report, 383 (Pl.), 431 (Chl.), 713 (Y.F.), 730 (R.F.)
Leake, C. D., with Anderson, 67, 851 (Misc.)
——, with Chen & Anderson, 851 (Misc.)
——, with David, 852 (Misc.)
Leão, A. E. de A., with da Fonseca, Jr., 404 (Der.)
Leblanc, L., (1032) (Mal.)
Le Bourdelles, A. C. B. & Henry, A. F. X., 140 (Mal.)
Le Bourdellès, B. & Liégeois, R., (611) (Mal.)
Le Chuiton & Négrié, 703 (Und.)

Le Chuiton, F., with Nicolle & Anderson, 731 *bis* (R.F.)
Leclerc, with Bablet & Joyeux, 245 (Rab.)
Le Cousse, (13) (Bl.)
Lee, C. U., 206 (Hel.)
Lee, P., with Cooley, 40 (Misc.)
Lee, S. W. T., 890 (S.S.), 942 *bis* (Bl.)
Lees, J. M., with Morgan, 479 (Oph.)
Lefrou, G., 865 (Misc.)
Le Gac, P., 173 (Hel.), 921 (S.S.), 957 (Lep.)
——, with Muratet, 304 (R.F.)
Le Gac, P. L., 661 (Sp.)
Leger, M., 70 (Misc.), 194 (Hel.), (296) (Y.F.), 352 (S.S.), 393, (395), 875 (Pl.), 602 (Mal.), 654 (Lep.), 971 (Oph.)
Legezynski, S. & Markowski, S., 256 (Rab.)
Leibly, F. J., 267 (Am.)
Leisermann, L. I., 136 (Mal.)
Lejeune, E., with van Nitsen, Miguens, Serra & Van den Branden, 439 (Y. & S.)
Lemaire, E., 70 (Misc.)
Lemaire, G., 625 (Fev.)
—— & Bardenat, E., 875 (Pl.)
Lemierre, A. & Rudolf, M., 948 (Bl.)
Lemoine, with Loygue, (740) (Lept.)
Lenfeld, J., 647 *bis* (K.A.)
Lengsfeld, W., 469 (Pel.)
Leonard, C. S., with Reiner, 359 (S.S.)
——, with —— & Chao, 359 (S.S.)
Lépine, P., 908 (S.S.)
——, with Levaditi, Ravaut & Schoen, 937, 938 (C.Bu.)
Le Prince, H., (296) (Y.F.)
Le Prince, J. A., 1027 (Mal.)
—— & Johnson, H. A., 80 (Misc.)
Leprosy Review, 323 *bis*, 651, 954 *bis* (Lep.)
Le Roux, J. J. du P., 325 (Lep.)
Le Roux, P. L., 195 (Hel.)
Le Roy des Barres, A., 118 (Mal.)
Lester, H. M. O., 520 (Z.)
——, with Taylor, 368 (S.S.)
Lestoquard, F., with Parrot & Donatien, 157 (K.A.)
de Lestrange, Y., with Fourneau, Tréfouel, M. & Mme., Stefanopoulo, Benoit & Melville, 64 (Misc.)
Le Sueur, E. & Hutchison, W., 409 (Der.)
Letonturier, Martin & Souchard, 40 (Misc.)
Levaditi, C., Anderson, T., Selbie, F. R. & Schoen, R., 302 (R.F.)
——, Bardet, J., Tchakirian, A. & Vaisman, A., 908 (S.S.)
—— & Po, L. Y., 305 (R.F.)
——, Ravaut, P., Lépine, P. & Schoen, R., 937, 938 (C.Bu.)
Levin, A. L., 206 (Hel.)
Levin, W., 701 (Und.)
Levinthal, W., 877 (Pl.)
Levy, A. S., with da Fonseca, Jr., 399 (Der.)
Levy, J., 42 (Misc.)
Lévy-Franckel, with Louste & Gadaud (658) (Lep.)
Lewillon, R., 42 (Misc.)
Lewis's Medical & Scientific Circulating Library, 698 (B.R.)
Lewthwaite, R., 251 (Rab.), 631, 633 (Fev.)
Liégeois, R., with Le Bourdellès, (611) (Mal.)
Lim, C. E. & Kurotchkin, T. J., 420 (Myc.)
——, with——, 421 (Myc.)
Lima A. da C., with Aragao, (296 *bis*) (Y.F.)
Limtscher, L. F. & Sankin, S. L., 673 (Hel.)
Lindberg, K., 876 (Pl.)
Linden, H., with Traum, 917 (S.S.)
Ling, W. P., 25 (Misc.)
Lingenfelter, J. S., with Hoffstadt, 421 (Myc.)
Lins, S. A., (296) (Y.F.)
Linton, R. W., 377 (S.S.)
Lipatova, T., with Goubarev, 877 (Pl.)
Lister, S., 557 (Lab.)
Liverani, E., 11 (Bl.)
Lloyd, R. B. & Napier, L. E., 514 (Z.)
Lobo, A., with Godoy & Cruz, Jr., 586 (Mal.)
Locatelli, P., 372, 375 (S.S.)
Loeper, M. & Tonnet, J., 679 (Hel.)
Löffler, E. & Schweinburg, F., 745 (Rab.)
Löffler, W. & v. Albertini, A., 86 (Und.)
Löhe, H. & Blümmers, K., 453 (C.Bu.)
Lokhov, M., with Bezsonova, 394 (Pl.)
London, J., 274 (Dys.)
Long, J. D., 382 (Pl.)
Longo, D., 113 (Mal.)
van Loon, F. H. G., 864 (Misc.)
Lopez, J. A., 81 (Misc.)
Lopez Nussa, E., 845 (Misc.)
Lo Presti-Seminerio, F., 645 (K.A.)
Loram, C. T., 766 (Misc.)
Lorando, N. & Chaniotis, N., 615 (Fev.)
Lorando, N. I., 132 (Mal.)
Lorando, W. J., (611) (Mal.)
v. Löte, J., 743 (Rab.)
Lotze, H., (552) (Z.)
Loucks, H. H., 204 (Hel.)
Lourie, E. M., 908 (S.S.)
——, with Adler & Theodor, 516 (Z.)
Louste, Lévy-Franckel & Gadaud, (658) (Lep.)
Low, G. C., 47 (Misc.), 450 (C.Bu.)
—— & Cook, A. B., (866) (Misc.)
—— & Dixon, D. S., 50 (Misc.)
—— & Fairley, N. H., 290 (Y.F.)
Loygue & Lemoine, (740) (Lept.)
Lubieniecki, H., 689 (Hel.)
de Luca, B., 130 (Mal.)
Lucca, A., 164 (K.A.)
Lucker, J. T., with Africa, 691 (Hel.)
Luiggi, F. J., 645 (K.A.)
Lukis, P. & Blackham, R. J., 869 (B.R.)
Lull, G. F., 885 (Chl.)
Luna, J. D., with Mazza, 165 (K.A.)
Lutrario, 125 (Mal.)
Lutz, A., 282 (Y.F.)
Lutz, H., 202 (Hel.)
Lutz, J. R., with Bassler, 660 (Sp.)
Lynch, K. M., 428 (B.R.)

M

Maass, E. & Vogel, H., 194 (Hel.)
MacArthur, W. P., 209 (Hel.)
MacCallan, A. F., 965, 967 (Oph.)
McCarthy, D. D., 21 (Misc.)
M'Cluskie, J. A. W., 317 (R.B.F.)
McCord, J. B., 766 (Misc.)
McCoy, G. W., 252 (Rab.)
McCoy, O. R., 238 (Hel.)
McCulloch, W. E., 555 (Lab.)
McDaniel, J. C., with Nutter, 978 (Mal.)
Macdonald, G., 995 (Mal.)
—— & Chowdhury, K. L., 987 (Mal.)

McDowall, R. J. S., 432 (Chl.)
McFadyen, A. A., 638 (K.A.)
MacFadyen, J. A., 271 (Am.)
Macfie, J. W. S., with Goodson & Henry, 849 (Misc.)
McGolrick, L., 844 (Misc.)
MacGregor, M. E., 504 (Z.)
McGusty, V. W. T., 400 (Der.)
Machattie, C., with Mills & Chadwick, 158 (K.A.)
Maciel, H., 454 (G.V.), 670 (Hel.)
McKendrick, A. G., 247 (Rab.)
McKenzie, A., 465 (Pel.)
Mackie, F. P., 45 (Misc.)
——, with Fairley & Jasudasan, 196 (Hel.)
McKie, M., with Burnet & Wood, 273 (Dys.)
McKinley, E. B., with Ashford & Dowling, 425 (Myc.)
—— & Douglass, M., 508 (Z.)
——, with Scott, 425 (Myc.)
Mackinnon, M., 35 (Misc.)
Maclean, G., 342, 343 (S.S.)
McLean, N., 122 (Mal.)
MacMahon, H. E. & Weiss, S., 66 (Misc.)
Macphail, N. P., 106, 978 (Mal.)
Madras, 971 (Oph.)
Maeji, Y., (801) (Am.)
Magalhaes, A. de G., 728 (Y.F.)
de Magalhães, O., 419 (Myc.)
Mahadevan, V. & Raman, T. K., 455 (Bb.)
Mahaffy, A. F., with Beeuwkes & Bauer, 283 (Y.F.)
Mahlangeni, 768 (Misc.)
Maisler, A., with Garnier & Nicaud, (740) (Lept.)
Maitra, G. C., with Tomb, 885 (Chl.)
Majumdar, A. R., 336 (B.R.)
Makel, H.P., 413 (Der.)
—— & Gilder, W., 12 (Bl.)
—— & de Vault, V. T., 221 (Hel.)
Malaguti, A., 87 (Und.)
Malaret, P. S., 107 (Mal.)
Malaria-Commissie uit den Gezondheidsraad, 116 (Mal.)
Malayan Medical Journal, 1028 (Mal.)
Maldonado Maldonado, A., 682 (Hel.)
Mallick, S. M. K., with Ahuja, 332 (Lep.)
Malone, R. H., with Asheshov, Taylor, Morison & Iyengar, 863 (Misc.)
Maltsberger, J. R., 107, 978 (Mal.)
Manai, A., with Franco, 149 (K.A.)
Manalang, C., 146, 587 (Mal.), 495, 512 (Z.)
Manca, S., 999 (Mal.)
Mandoul, H., (395) (Pl.)
Mandry, O. C., (280) (Dys.)
Mangiola, M., 687 (Hel.)
Manifold, J. A., 840 (Misc.), 1007 (Mal.)
Mann, F. C., with Essex & Markowitz, 542 (Z.)
Manoussakis, E., 64 (Misc.), 619 (Fev.)
Mansell, R. A., 987 (Mal.)
Manson-Bahr, P., 8 (Bl.), 47 (Misc.), 925 (S.S.), 1005, 1010 (Mal.)
Manteufel, P. & Herzberg, K., 729 (Y.F.)
—— & Taute, M., 427 (B.R.)
Manwell, R. D., 600 (Mal.)
Maplestone, P. A., 204, 207, 230 (Hel.)
Marcandier & Bideau, 97 (Fev.)
Marcandier, A., with Plazy, 624 (Fev.)
Marchiafava, E., 587 (Mal.)
Marchoux, E., 852 (Misc.)
—— & Chorine, V., 495 *bis* (Z.), 738 (R.F.)
——, Markianos, J. & Chorine, V., 656 (Lep.)
Marçon, L. & Marçon, H., 631 (Fev.)
Marie, A. C. & Urbain, A., 256, 746 (Rab.)
Mariotti-Bianchi, with Gabbi, 26 (Misc.)
Markianos, J., 652, 657 (Lep.)
——, with Marchoux & Chorine, 656 (Lep.)
Markowitz, J., with Essex & Mann, 542 (Z.)
Markowski, S., with Legezynski, 256 (Rab.)
Marmo, A., (307) (R.F.), 860 (Misc.)
Marmorston-Gottesman, J., with Perla, 492 (Z.)
——, —— & Vorzimer, J., 492 (Z.)
Marone & Tessitore, C., 845 (Misc.)
de Marqueissac, 345 (S.S.)
Marques, A., 237 (Hel.)
Marsh, F., 888 (H.S.)
Marshall, M. S. & Jared, D., 89 (Und.)
Martin, with Letonturier & Souchard, 40 (Misc.)
Martin, C. J., 888 (H.S.)
Martin, J. F., with Paviot & Dechaume, 87 (Und.)
Martin, D. L., 270 (Am.)
Martínez Vinueza, J. J., 385 (Pl.)
Martini, E., 500 (Z.)
—— & Achundow, I., 508 (Z.)
Marzinowsky, E. I., 620 (Fev.)
Masen, J. M., with Vedder, 1002 (Mal.)
Mason, M., 169 (B.R.)
Masselot, F., 699 (Und.)
Massia, G., with Nicolas & Weigert, (658) (Lep.)
Massias, C., 135 (Mal.), 528 (Z.)
Masslow, A. W., 513 (Z.)
Massoud, F., 480 (Oph.)
Mata Merchán, M. & Mata Merchán, J., 645 (K.A.)
Mathieu, 682 (Hel.), 951 (Bl.)
Mathieu, H., 12 (Bl.)
Mathis, C. & Durieux, C., 303, 735 (R.F.)
Matossi, R., 223 (Hel.)
Matsuura, T., with Morishita, Namikawa & Tanikawa, (1032) (Mal.)
Mattei, C., 789 (Am.)
di Mattei, G., 82 (Misc.)
Maurice, G. K., 343, 379 (S.S.)
Maxwell, J. L., 673 (Hel.)
Maxwell, J. S., (147) (Mal.)
Mayer, T. F. G., 320 *bis* (Lep.)
Mazet, M., 627 (Fev.)
Mazza, S., 305 (R.F.), 483 (Z.)
—— & Luna, J. D., 165 (K.A.)
——, Romaña, C. & Schürmann, K., 923 (S.S.)
Medulla, C., 93, 99 (Fev.), (147) (Mal.), 701 (Und.), 732 (R.F.)
Megaw, J. W. B., 31 (Misc.)
Megaw, J. W. D., 623 (Fev.)
de Meillon, B., 514 (Z.)
Mélanidi, C. & Stylianopoulo, M., 617 (Fev.)
Mello, A. da S., (280) (Dys.)
de Mello, F., (147) (Mal.), 809 (Hist.)
——, Brás de Sá, L J. & d'Abreu, M., 1008 (Mal.)
de Mello, I. F., 955 (Lep.)
Melnotte, P., 594 (Mal.)
Melville, K. I., with Fourneau, Tréfouel, M. & Mme., Stefanopoulo, Benoit & de Lestrange, 64 (Misc.)

Menaut, 960 (Lep.)
Mendelson, R. W. & Beam, M. P., 662 (Sp.)
Mendis, J. C., with Richmond, 105 (Mal.)
de Mendonça, F. C., 799 (Dys.)
Menk, W., 733 (R.F.)
Mennonna, G., 799 (Dys.)
Menon, K. N., with Taylor, 248 (Rab.)
Menon, K. P., with King, Pandit & Iyer, 211 (Hel.)
Menon, T. B., 279 (Dys.)
Mer, G., with Kligler, 589, 599, 1008 (Mal.)
Mertens, W. K., 621 (Fev.)
Merzbacher, L., 118 (Mal.)
Mesnard, J. & Bordes, L. A., 580 (Mal.)
—— & Morin, H., 1019 (Mal.)
Mesnil, F., 379 (S.S.)
Messimy, R., 412 (Der.)
Mestre, I. J., with Castello, 414 (Der.)
Metz, M., 881 (Chl.)
Meyer, J., with Sartory & Meyer, M., 424 (Myc.)
Meyerhof, M., 480 (Oph.)
Meyers, F. M., 844 (Misc.)
Mhaskar, K. S. & Caius, J. F., 68 (Misc.)
——, with —— & Isaacs, 68 (Misc.)
Michailow, S., with Latko, 938 (C.Bu.)
Michel, R., with Bailly, 257 (Rab.)
Micheletti, E., 133 (Mal.)
Middleton, W. S., 819 (Hist.)
de Miglio, U., (611) (Mal.)
Miguens, with van Nitsen, Lejeune, Serra & Van den Branden, 439 (Y. & S.)
Miguens, J., 440 (Y. & S.)
Mikaelian, R. C., 968 (Oph.)
Milani, C., with Cuboni, (1031) (Mal.)
Milhaud, with Piéry, 26 (Misc.)
Milian & Degos, R., (658) (Lep.)
Milio, G., 164 (K.A.)
Miller, H. M., Jr., 205, 679 (Hel.)
Miller, J. W., 922 (S.S.)
——, with d'Aunoy, 251 (Rab.)
Milliken, G., with Chandler & Schuhardt, 217 (Hel.)
Mills, E. A. & Machattie, C., with Chadwick, C. R., 158 (K.A.)
Minami, S. & Ehara, I., 215 (Hel.)
—— & Sato, T., 183 (Hel.)
Minervin, S. M., 882 (Chl.)
Mirra, G., (13) (Bl.)
Mirza, M. B., 221 (Hel.)
Missiroli, A., 607, 609 (Mal.), 835 (Misc.)
——, with Hackett, 569 (Mal.)
Mitamura, T., with Nagayo, Tamiya & Sato, 635 (Fev.)
Mitchell, J. A., with Pirie, J. H. H., Rhodes, W. F. & Powell, W., 391 (Pl.)
Mitra, S., (692) (Hel.)
Miyagawa, Y. & Okada, R., 238 (Hel.)
Miyajima, M., 536 *bis* (Z.)
Miyamoto, S., 509 (Z.)
Miyao, I., 446 *ter*, 448 (Y. & S.)
——, with Hasselmann, 413 (Der.)
——, with Schöbl & Tanabe, 443 (Y. & S.)
Miyoshi, S., with Tôyama & Kubokawa, 332 (Lep.)
Mizushima, H., with Tsuchiya, 488 (Z.)
Mochkovski, C., 996 (Mal.)
Mochtar, A., 319 (R.B.F.)
Moeller, J., 763 (Misc.)
Mohammed, A. S., 669, (692) (Hel.)
Molinelli, E. A. & Ré, P. M., 332 (Lep.)
Mollow, W., 205 (Hel.)
Moltschanow, S. A., with Burowa, 154 (K.A.)
Momma, K., 209 (Hel.)
Monaldi, T. de S., with Ninni, 963 (Lep.)
Moncarey, 11 (Bl.)
Monier, H. M., 1012 *quat.* (Mal.)
Monier, H., with Lavergne, 1022 (Mal.)
Monteiro, J. L., 724 (Y.F.)
Monteleone, R., 595 (Mal.)
Montgomery, H., 545 (Z.)
Moore, D. F., 38 (Misc.)
Moore, D. G. F., 478 (Oph.)
Moore, J. H., with Clemesha, 576 (Mal.)
Moore, J. J., 48 (Misc.)
Moraleda, C., with De la Camara, 601, 1011 (Mal.)
Morax & Nida, 968 (Oph.)
Morax, V., 476, 477, 968 (Oph.)
Moreau, 873 (Pl.)
Moretti, G., 226 (Hel.)
Morgan, E. L., 616 (Fev.)
Morgan, L. S., 638 (K.A.)
Morgan, O. G. & Lees, J. M., 479 (Oph.)
Morin, H., with Mesnard, 1019 (Mal.)
Morin, H. G. S., 112, 580, (1032), (Mal.)
Morishita, K., 498 (Z.), 1009 (Mal.)
—— & Namikawa, H., 1010 (Mal.)
——, ——, Matsuura, T. & Tanikawa, K., (1032) (Mal.)
——, with Oda & Namikawa, 1009 *bis* (Mal.)
Morison, J., 560 (Lab.)
——, with Asheshov, Taylor, Malone & Iyengar, 863 (Misc.)
Morrell, C. A., with Wakeman, 13 (Bl.), 293, 722 (Y.F.)
Morrison, A., (878) (Pl.)
Morrison, H., 555 (Lab.)
Moufel, P. P., 120 (Mal.)
Mouillac, 771 (Misc.)
Mouquet, A., 449 (Y. & S.)
Mtschedlidse, I., with Kalandadse, 539 (Z.)
Mtschedlidzé, J., 674 (Hel.)
Mu, J., with Frazier & Kurotchkin, 418 (Myc.)
Mühlens, P., 594 (Mal.)
—— & Knabe, K., 947 (Bl.)
——, with Nocht, (611) (Mal.)
Muir, E., 158 (K.A.), 958 (Lep.)
Mukherjee, S. C., 434 (Chl.)
Müller, H. R. & Stender, A., 191 (Hel.)
Mulligan, H. W., with Sinton, 146 (Mal.)
Munford, S. A., 467 (Pel.)
Murakawa, G., 916 *quin.* (S.S.)
Murat, with Audibert, 100 (Fev.)
Muratet, L. & Le Gac, P., 304 (R.F.)
Muraz, G., 352 (S.S.)
Murgatroyd, F., with Yorke, 350 (S.S.)
——, with —— & Hawking, 909, 910 (S.S.)
——, with —— & Owen, 1 (Bl.)
Murphy, R. A., (147) (Mal.)
Murugiah, C., 319 *bis* (R.B.F.)
Musger, 451 (C.Bu.)
Mynssen, G. E. H. V., 137 (Mal.)

N

Nag, S. C., 65 (Misc.), 179 (Hel.)
Nagano, K., 169 (Hel.)

Nagayo, M., Tamiya, T., Mitamura, T. & Sato, K., 635 (Fev.)
Nagoya, T., 201 (Hel.)
Nair, K. G. & Raju, S., (13) (Bl.)
Naito, K., 691 (Hel.)
Nájera, L., 355, 903, (925 *bis*) (S.S.)
Nakajima, G., 276 (Dys.)
Namikawa, H., (866) (Misc.)
——, with Morishita, 1010 (Mal.)
——, with ——, Matsuura & Tanikawa, (1032) (Mal.)
——, with Oda & Morishita, 1009 *bis* (Mal.)
Nanavati, B. P., with Duggan, 479 (Oph.)
Napier, L. E., 515 (Z.), 693 (B.R.)
—— & Gupta, C. R. D., 160 (K.A.)
—— & Haldar, K. C., 161 (K.A.)
——, with Lloyd, 514 (Z.)
Narayanin, C., with Labernadie, (438) (Chl.)
Nash, T. A. M., 520 (Z.)
Nattan-Larrier, L. & Noyer, B., 371 (S.S.)
—— & Richard, L., 874 (Pl.)
Nature, 639 (K.A.)
Nauck, E. G., (242) (Hel.)
——, with Peña Chavarria, 54 (Misc.)
Naumann, H. E., 936 (C.Bu.)
Nayar, K. K., with Wright, 478 (Oph.)
Nazareth, F. X. de S., 843 (Misc.)
Neer, L. C., 748 (Rab.)
Neff, E. A., 321 (Lep.)
Negishi, K., (242) (Hel.)
Négrié, with Le Chuiton, 703 (Und.)
Neiva, C., 90 (Und.)
Nélis, P., with Van den Branden & Dumont, 305 (R.F.), 900 (S.S.)
Nelson, R. L., 271, 796 (Dys.)
Neri, F., 145 (Mal.)
Nesturch, M., with Grigorowa, 218 (Hel.)
Neuber, E., 218 *ter* (Hel.)
Neuda, P., (738) (R.F.)
Neumann, C. Z., 150 (K.A.)
Neumann, J., 967 (Oph.)
Neveu, R., 607 (Mal.)
Newell, W. G., with Kirby, 969 (Oph.)
Nguyen-Duy-Ha, with Haslé, 138 (Mal.)
——, with Normet & Hasle, 407 (Der.)
Nicaud, P., with Garnier & Maisler, (740) (Lept.)
Nicol, W. D., with James & Shute, 973 (Mal.)
Nicolas, J., Massia, G. & Weigert, H., (658) (Lep.)
Nicolau, C. T. & Gingold, N., 998 (Mal.)
Nicolau, S. & Kopciowska, L., 247 *bis* (Rab.)
——, Viala, J. & Kopciowska, L., 246 (Rab.)
Nicolle, C., 538 (Z.)
—— & Anderson, C., 307, 731 (R.F.)
——, —— & Le Chuiton, F., 731 *bis* (R.F.)
Nida, with Morax, 968 (Oph.)
Nieschulz, O., 340 (B.R.)
—— & Hofkamp, H. S., 222 (Hel.)
Nieuwenhuis, A. W., 986 (Mal.)
Nigeria, 554, 555 *ter* (Lab.)
Nikanorov, S., with Tikhomirova, 393 (Pl.)
Nikolsky, A., 117 (Mal.)
Ninni, C. & Monaldi, T. de S., 963 (Lep.)
Nishimura, H., 387 (Pl.)
——, with Ando & Kurauchi, 383 (Pl.)
Nishiyama, I., 183 (Hel.)
——, with Eguchi, 203 (Hel.)
van Nitsen, R., 763 (Misc.), 929 (Y. & S.)
——, Lejeune, E., Miguens, Serra, G. & Van den Branden, F., 439 (Y. & S.)
Nitzulescu, J., with Puscariu, 968 (Oph.)
Nocht, B., 4 (Bl.)
—— & Mühlens, P., (611) (Mal.)
Nolasco, J. O., 328, 329, 959 (Lep.)
Nöller, W., 781 (B.R.)
Nopoff, R., with Dios, 534 (Z.)
Normet, L., Hasle, G. & Nguyên-Duy-Ha, 407 (Der.)
Noronha, A. J., 497 (Z.)
de Noronha, R., 1010 (Mal.)
Norrie, F. H. B., 422 (Myc.)
Norris, B., 1027 (Mal.)
North, E. A., 333 (Lep.)
Nossina, V., with Schourenkova, 497 (Z.)
Noto-Soediro, R., (242) (Hel.)
Nowosselsky, W. A., (866) (Misc.)
Noyer, B., with Nattan-Larrier, 371 (S.S.)
du Noyer, M. R., with Langeron, 696 (B.R.)
Nudelman, M., 165 (K.A.)
Nuttall, G. H. F., 534 (Z.)
Nutter, R. B. & McDaniel, J. C., 978 (Mal.)
Nyasaland Protectorate, 498 (Z.)
Nye, L. J. J., 54 (Misc.)

O

Ochi, S., 184 (Hel.)
Ochoterena, I., 684 *bis* (Hel.)
O'Conner, F. W., 681 (Hel.)
——, Golden, R. & Auchincloss, H., (692) (Hel.)
O'Connor, M. H., (740) (Lept.)
Oda, T., Morishita, K. & Namikawa, H., 1009 *bis* (Mal.)
Odessaer Medizinische Zeitschrift, 175 (Hel.)
Odriosola, R., (552) (Z.)
O'Farrell, W. R., 200 (Hel.)
O'Flynn, J. A., (1032) (Mal.)
Ogata, N., with Ishiwara, 104 (Fev.)
Ogawa, S., with Takahashi, Ishikawa & Ida, 467 (Pel.)
Ohta, K., 458 (Bb.)
—— & Izumita, T., 459 (Bb.)
Oiso, T., 240 (Hel.)
Okada, R., with Miyagawa, 238 (Hel.)
Okano, M., 179 (Hel.)
Okonogi, T., 849 (Misc.)
Olitsky, P. K., 476 (Oph.)
——, Knutti, R. E. & Tyler, J. R., 966 (Oph.)
—— & Tyler, J. R., 965 (Oph.)
Olmer, 99, 624 (Fev.)
Olmer, D., 627 (Fev.)
—— & Olmer, J., 625 (Fev.)
Olpp, G., 810 (Hist.)
O'Malley, C. C., 478 (Oph.)
Omar, A. R., 394 (Pl.)
Onnizew, P., 1031 (Mal.)
Onorato, R., 769 (Misc.)
Onslow, D. V., 667 (Hel.)
Onsy, A., 186 (Hel.)
Ontaneda, L. E., with Gonzalez & Vidaurreta, 162 (K.A.)
Oonvala, J. H., 837 (Misc.)
O'Reilly, B. C. N., 440 (Y. & S.)
Orenstein, A. J., 192 (Hel.), 766 (Misc.)

Orlowa, A. A. & Schachow, S. D., 514 (Z.)
Orpen, L. J. J., 841 (Misc.)
Osborne, C. J., with Holder & Sommermeyer, 791 (Am.)
Osman, A. A., 34 (Misc.)
Ota, M., 543 (Z.)
—— & Sato, S., 962 *bis* (Lep.)
Otto, G. F., (242) (Hel.)
—— & Spindler, L. A., 174 (Hel.)
Otto, R., 551 (Z.)
Ottolenghi, D., 611 (Mal.)
Ouchakov, V., 250 (Rab.)
Owen, D. U., 642 (K.A.)
——, with Yorke & Murgatroyd, 1 (Bl.)
Owen, D. V., with Yorke, 606 (Mal.)
Owtscharenko, E. P., 549 (Z.)
Ozawa, M., 671 (Hel.)

P

Palawandow, H. B., 749 (Rab.)
—— & Serebrennaja, A. I., 741 (Rab.)
—— & Serebrjenaja, A. I., 245 *bis* (Rab.)
Paldrock, A., 330, 654 *bis*, (658) (Lep.)
Pallary, P., 544 (Z.)
Palmerlee, C. A., 686 (Hel.)
Palmowitch, S. M., with Remlinger & Bailly, 745 (Rab.)
Pamboukis, G., 618 (Fev.)
Pampana, E. J., 737 (R.F.)
Panayotatou, A., 159 (K.A.), 421, 423 (Myc.), 787, 790 (Am.)
Pandit, with Russell & King, 381 (Pl.)
Pandit, C. G., with King, Menon & Iyer, 211 (Hel.)
——, Pandit, S. R. & Iyer, P. V. S., (242) (Hel.)
Paneth, O., 320, 959 (Lep.)
de Paoli, P., 298 (R.F.), 407 (Der.), 751 (Rab.)
Papaïoannou, A., with Blanc & Joannidès, 404 (Der.)
Papanastasiou, E., (148) (Mal.)
Papert, J. L., 526 (Z.)
Paradiso, F., 154 (K.A.)
Paradoksow, L. F., 175 (Hel.)
Paras, E. M., 655 (Lep.)
Pardo-Castello, V. & Caballero, G. M., 653 (Lep.)
Parjono, Radsma, W. & Joenoes, M., 862 (Misc.)
Parodi, S., 422 (Myc.)
Parr, T., (242) (Hel.)
Parrot, L., 517 (Z.)
——, Donatien, A. & Lestoquard, F., 157 (K.A.)
——, with Sergent, Edm., Sergent, Et., Foley, Catanei & Senevet, 984 (Mal.)
Parsons, R. P., 807 (Hist.), 868 (B.R.)
Pascheff, 476 (Oph.)
Pastor Botija, F., with Lazaro Caballero, 164 (K.A.)
Patanè, C., 532 (Z.)
Paterson, A. R., (148) (Mal.)
Patriarca, M., (801) (Am.)
Patton, C. R., 48 (Misc.)
Paulson, M., with Andrews, 485 (Z.), 785 (Am.)
Paviot, J., Martin, J. F. & Dechaume, J., 87 (Und.)
Pavlovsky, E. N., (552) (Z.)
Pawan, J. L., 739 (Lept.), 1025 (Mal.)
—— & Camps-Campins, J., 863 (Misc.)
Pawlowsky, E. N., 175 (Hel.)
—— & Stein, A. K., 534 (Z.)
Pazienza, M., 405 (Der.)
Pearce, L., 426 (B.R.)
Pédaros, D., Caragianopoulos, G. & Valsamaki, A., 628 (Fev.)
Pedro, A., with Vianna, 457 (Bb.)
Peill, S. G., (242) (Hel.)
Peirier, with Advier, 330 (Lep.)
Peltier, 575, 975 (Mal.)
Peña, R., 425 (Myc.)
Peña Chavarria, A. & Nauck, E. G., 54 (Misc.)
——, Serpa, R. & Bevier, G., 281 (Y.F.)
Penington, R. G., 950 (Bl.)
Penning, C. P. J., 822 (Hist.)
Penso, G., 222, 687 (Hel.), 263, (801) *bis* (Am.)
Penteado, J., with Vellard, 551 (Z.)
Pereira, Jr., 423 *bis*, 425 (Myc.)
Perepérez, F., 645 (K.A.)
Péretz, H., 480, 969 (Oph.)
Perez, M., 514 (Z.)
Pergher, J., 900 (S.S.)
Perla, D. & Marmorston-Gottesman, J., 492 (Z.)
——, with—— & Vorzimer, 492 (Z.)
Perry, H. M., 276 (Dys.)
Peruzzi, M., 800 *bis* (Dys.)
Pessôa, S. B., 833 (Misc.)
Peter, F. M., 187 (Hel.)
Peterson, C. H., with Archer, 688 (Hel.)
Petridis, P., (801) (Am.)
Petrov, V. P., 94 (Fev.)
Petrowsky, I. N., with Stradomsky, Popow & Rudnew, 142 (Mal.)
Pettit, A., 716 (Y.F.)
Petzetakis, 265, (801) (Am.)
Peverelli, P., 96 (Fev.)
Phelps, B. M., 107 (Mal.)
Phelps, J. R., Smith, O. A., Carroll, H. H., Washburn, W. A. & Beagley, K. E., 215 (Hel.)
Philaire, 137 (Mal.)
Philip, C. B., 292 (Y.F.)
Philip, C. R., 585 (Mal.)
Philippine Journal of Science, 649 (Lep.)
Philiptschenko, A. A., 261 *bis* (Am.), (280) (Dys.)
Phipson, E. S., 45 (Misc.)
Phisalix, M., 752 (Rab.)
Photakis, B. A., 394 (Pl.)
Photinos, G. T., 161 (K.A.)
Photinos, P. B., 646 (K.A.)
Pichat, J., (296) (Y.F.)
Pieri, J., with Boinet, 626 (Fev.)
——, with Joyeux, 630 (Fev.)
—— & Raybaud, A., 627 (Fev.)
Piéry & Milhaud, 26 (Misc.)
Pijper, A. & Dau, H., 101, 636 (Fev.)
Pikul, J., 156 (K.A.)
Pinelli, L., 141 (Mal.)
Pinto, C. & da Fonseca, F., 527 (Z.)
Pinto, S. C. F., with de Andrade, (146) (Mal.)
Pirami, E., with Franchini, 643 (K.A.)

Pirgialis, A., 628 (Fev.)
Pirie, J. H. H., 391 (Pl.)
Pirngadi, R., with Heinemann & Koumans, 30 (Misc.)
Pirot, R., 616 (Fev.)
Plaut, F. & Grabow, C., 301 (R.F.)
Plazy, 820 (Hist.)
Plazy, L. & Marcandier, A., 624 (Fev.)
Po, L. Y., with Levaditi, 305 (R.F.)
Podjapolskaja, W. P., with Skrjabin, 675 (Hel.)
Podjapolskaya, W. P., with —— & Schichobalowa, 208 (Hel.)
Podyapolskaya, B. P., with Scriabine & Statirova, 182 (Hel.)
Pontano, T., 620 (Fev.)
Popoff, with Couvy, 386 (Pl.), 839 (Misc.)
Popoff, S., with Assali, 385 (Pl.)
Popow, W., 877 (Pl.)
Popow, W. W., with Stradomsky, Petrowsky & Rudnew, 142 (Mal.)
Pordon, A. A. A. C., 861 (Misc.)
Porter, A., 232 (Hel.), 545 (Z.)
Portugal, H., 158 (K.A.)
Postmus, S., (280) (Dys.)
Poston, M. A., with Amoss, 89 (Und.)
Pou, R. P., 307 (R.F.)
Powell, A. T. W., 272 (Dys.)
Powell, W., 391 (Pl.)
Power, R. W. & Johnston, H. W., 226 (Hel.)
Pozzi, A., 139 (Mal.)
Pradhan, Y. M., 220 (Hel.)
Prado, A. & Carvalho, A. E., 114 (Mal.)
Pratti, 891 (S.S.)
Prawirohardjo, R. S., (692) (Hel.)
Prebil, M., (280) (Dys.)
Predtetschenskaja, L. A., with Glusmann & Solowjowa, 244 (Rab.)
Preyer, G., 318 (R.B.F.)
Prieur, M., with Launoy, 362 *bis*, 906 (S.S.)
Proceedings of the Celebration of the Three Hundredth Anniversary of the First Recognized Use of Cinchona, 1033 (B.R.)
Proceedings of the Royal Society of Medicine, 31 (Misc.)
Procter, R. A. W., 113 (Mal.), 762 (Misc.)
Progrès de l'Aluminium, 777 (B.R.)
Provost, J., with Sicé, Boisseau & Deniel, 901 (S.S.)
Puntigam, F., 143 (Mal.)
Puntoni, V., 419 (Myc.)
Purdy, J. S., 834 (Misc.)
Puscariu, E. & Nitzulescu, J., 968 (Oph.)
Puymartin, C., with Laignel-Lavastine & Boquien, (740) (Lept.)

Q

Quenardel & Kimsan, (801) (Am.)
Quinnell, E., with Ashford, 699 (Und.)
Quiyum, M. A., 50 (Misc.)

R

Rabello, E., 414 (Der.)
Radsma, W., 73, 74 (Misc.)
——, with Parjona & Joenoes, 862 (Misc.)
Radosavljević, A., 603 (Mal.)
Ragain, L., 965 (Oph.)
Rajewski, A. S., 333 (Lep.)
Rajewsky, A. S., (658) (Lep.)
Raju, R. B. V. G. & Sircar, B. L., (438) (Chl.)
Raju, S., with Nair, (13) (Bl.)
Raju, V. G., 435 (Chl.)
—— & Sarkar, A. K., 885 (Chl.)
Rakshit, A., with Dhur-Roy, (658) (Lep.)
Raman, T. K., with Mahadevan, 455 (Bb.)
Ramos y Silva, J., 400 (Der.)
Rampon, L., 157 (K.A.)
Ramsay, G. C., 579 *bis* (Mal.)
Ramsay, G. W. St. C., 555 (Lab.)
Ramsey, G. H., 714 (Y.F.)
Randria, S., 393 (Pl.)
Raney, R. B., (280) (Dys.)
Rangoon, 560 (Lab.)
Ranjeva, J. B., 174 (Hel.)
Rao, K. R., 577 (Mal.)
Rao, S. S., 171, 212, 683 (Hel.)
——, with Acton, 212, 213 *bis*, 682 (Hel.)
——, with Chopra & Choudhury, 216 (Hel.)
—— & Iyengar, M. O. T., 212 (Hel.)
Rasoamanana, G., 875 (Pl.)
Rastegaieff, E. F., with Yakimoff, 306 (R.F.)
Ratner, M. I., with Kritschewski & Semzova, 855 (Misc.)
Ravaut, P., with Levaditi, Lépine & Schoen, 937, 938 (C.Bu.)
Raybaud, A., 100, 627 (Fev.), 140 (Mal.), (307) (R.F.)
——, with Pieri, 627 (Fev.)
Raymond-Hamet, (280) (Dys.)
Raynal, J., 670 (Hel.)
Ré, P. M., with Molinelli, 332 (Lep.)
Read, H., 688 (Hel.)
Records of the Malaria Survey of India, 105, 577 (Mal.)
Reddingius, T., 549 (Z.)
Reed, A. C., 468 (Pel.), 790 (Am.)
Regendanz, P., 374, 918, (925) (S.S.)
Rehbein, M., 1011 (Mal.)
Reimann, H. A., with Chung, 643 (K.A.)
Reiner, K. & Leonard, C. S., 359 (S.S.)
——, —— & Chao, S. S., 359 (S.S.)
Remlinger, P., 75 (Misc.), 255 (Rab.)
—— & Bailly, J., 243, 250, 254, 255, (257 *bis*), 741 *bis*, 742 *bis*, 744, 747, 750, 751 (Rab.), 303, 304 (R.F.)
——, Palmowitch, S. M. & Bailly, J., 745 (Rab.)
Remontet, E., with Vialatte, 140 (Mal.)
Renaud, (692) (Hel.)
Rennie, D. C., 887 (H.S.)
Revue de Médecine et d'Hygiène Tropicales, 159 (K.A.), 787 (Am.)
Rey, J. C., with Dassen, (241) (Hel.)
Reynolds, F. H. K., with St. John & Simmons, 507 (Z.), 613 (B.R.)
——, with Simmons & St. John, 114 (Mal.)
Reyntjes, E. J., with Walch & van Breemen, 1028 (Mal.)
Rezende, M. & Sobrinho, J. M., 936 (C.Bu.)
Rho, F., 57, 839 (Misc.), (729) (Y.F.)
Rhodes, W. F., 391 (Pl.)
Rice, E. M., 986 (Mal.)
Richard, L., with Nattan-Larrier, 874 (Pl.)
Richardson, C. H. & Shepard, H. H., 509 (Z.)

Richmond, A. E. & Mendis, J. C., 105 (Mal.)
Ridder, 800 (Dys.)
Riding, D., 272, 797 (Dys.)
Ridlon, J. R., 315 (Lept.)
Riegl, R., with Tsykalas, 190 (Hel.)
Riley, W. A., 115 (Mal.)
Rille, J. H., 474 (Pel.)
Rinehart, J. F. & Anderson, H. H., 850 (Misc.)
Riou, with Toullec, (964) (Lep.)
Rjabowa, M., with Skliar, 594 (Mal.)
Robert-Lévy, with de Lavergne & Kaiser, 311 (Lept.)
Roberts, C. E., 942 (Bl.)
Roberts, M. A. W., 66 (Misc.)
Robertson, A., 317 (R.B.F.), 378 (S.S.)
Robertson, E. A., 231 (Hel.)
Robertson, R. L., 48 (Misc.)
Robic, J., 35 (Misc.)
Rocca, G. C., with Dominici, 145 (Mal.)
Rocchi, F., 1021 (Mal.)
Rocha, F., (1032) (Mal.)
da Rocha, M., (801) (Am.)
Rockefeller Foundation, 168 (B.R.)
Rodecurt, M., 491 (Z.)
Rodenwaldt, E. R. K. & Cohen, A. J., 85 (Und.)
Röder, R., 1034 (B.R.)
Rodhain & Houssiau, F., 219 (Hel.)
Rodhain, J., 689 (Hel.)
Rodriguez, J. N., 474 (Pel.)
Rodriguez Lopez-Neyra, C., (242) (Hel.)
Roe, J. H., with Whitmore, 6 (Bl.), 107 (Mal.)
Roesle, E., 29 (Misc.)
Roest, P. K., (866) (Misc.)
Rogers, L., 38, 39, 856 (Misc.), (658) (Lep.)
Rogers, L. M., with Goldberger, Wheeler & Sebrell, 473 (Pel.)
Romaña, C., with Mazza & Schürmann, 923 (S.S.)
Rombach, K. A., (148) (Mal.)
Romeo, N., 203 (Hel.)
Ronnefeldt, F., (611) (Mal.), 790 (Am.), 861 (Misc.)
de Rook, H., with Brug, 217 (Hel.)
—— & Soesilo, R., (552) (Z.)
da Rosa, A. F., with da Fonseca, Jr., 401 (Der.)
Rosa, B., 751 (Rab.)
Rosenthal, S. M. & Voegtlin, C., 60 (Misc.)
Roskin, G., 911, 912 (S.S.)
Roskott, E. R. A. L., 771 (Misc.)
Ross, F., 48 (Misc.)
Ross, G. A. P., 766 (Misc.), (1032) (Mal.)
Ross, R., 814 (Hist.)
Roubaud, E., 506 (Z.)
—— & Toumanoff, C., 507, 509 (Z.)
Roudinesco, with Sézary, 657 (Lep.)
Rousset, J., with Doubrow, 234 *bis* (Hel.)
——, with Garin & Gonthier, 233 (Hel.)
Roux, P., with Buchanan, 796 (Dys.)
Row, R., 145, 1023 (Mal.), 155 (K.A.)
Row, R. S. T. M., with Dikshit, 959 (Lep.)
Roy, A., 655 (Lep.)
Roy, A. C., with Boyd, 51 (Misc.), 216 (Hel.)
Roy, D. N. & Chowdhury, K. L., 112 (Mal.)
——, with Strickland, 1018 (Mal.)
Rubinato, G., (103) (Fev.)
Rubino, C., 961 (Lep.)
Rubino, M. C., 960 *bis* (Lep.)
Rubinstein, P. L., 71 (Misc.)
—— & Golubewa, E. E., 70 (Misc.)
Rudnew, G. P., with Akulowa, 388 (Pl.)
—— & Krumberg, A. J., 703 (Und.)
——, with Stradomsky, Petrowsky & Popow, 142 (Mal.)
——, with Tinker, 390 (Pl.)
Rudolf, M., with Lemierre, 948 (Bl.)
Ruffino, P., with Spoto, 957 (Lep.)
Ruiz, F. R. & Fotheringham, W. T., 452 (C.Bu.)
Rukke, G. V., 878 (Pl.)
Rule, A. M., with Kolmer, 907 (S.S.)
Russell, King & Pandit, 381 (Pl.)
Russell, H., 730 (R.F.)
——, with Hawe, 244 (Rab.)
Russell, P. F., 1011 (Mal.)
Russo, C., 384 (Pl.)
Rustomjee, K. J., 610 (Mal.)
Ruttledge, W., 534 (Z.)
Rybinsky, S. B., 989 (Mal.)
Ryrie, C. A., 669 (Hel.)

S

Sabolotnow, P. & Schmidt, B., 394 (Pl.)
Sabrazès, J., 652 (Lep.), (801) (Am.)
Sadler, E. S., Dilling, W. J. & Gemmell, A. A., 848 (Misc.)
Sadowsky, I. J., with Głusman & Solowjewa, 244 (Rab.)
Sagel, W., 300 (R.F.)
Sainte-Marie, P. E. F., with Vialatte, 994 (Mal.)
St. John, J. H., Simmons, J. S. & Reynolds, F. H. K., 507 (Z.)
——, with —— & ——, 114 (Mal.), 613 (B.R.)
Saito, J., 534 (Z.)
Saldanha, J. L., with Gharpure, 783 (Am.)
Salisbury, E. I. & [Corrigan, J. A.], 107, (Mal.)
Samson, J. G., Lara, C. B. & Cruz, M. C. 329 (Lep.)
Sanders, E. P., with Cleveland, 791, 792 (Am.)
Sanders, J., (148) (Mal.)
Sanfilippo, E., 704 (Und.)
Sanguinetti, L. V., (801) (Am.)
Sankin, S. L., with Limtscher, 673 (Hel.)
Sanner, 52 (Misc.)
Sargent, W. S., 447 (Y. & S.)
Sarkar, A. K., with Raju, 885 (Chl.)
Sarkar, S. L., 165 (K.A.), (611), (1032) (Mal.)
Sarkisian, A. B., 605 (Mal.)
Sarramon, 434 (Chl.)
Sartory, A., Meyer, M. & Meyer, J., 424 (Myc.)
Sassykina, T., with Iwanowsky, 391 *bis* (Pl.)
Sathe, R. G., 388 (Pl.)
Sato, K., with Nagayo, Tamiya & Mitamura, 635 (Fev.)
Sato, S., with Ota, 962 *bis* (Lep.)
Sato, T., with Minami, 183 (Hel.)
Sautet, J., 172 (Hel.), (866) (Misc.)
Sawhney, M. R., 475 (Oph.)
Sayers, E. G., 46 (Misc.)
Scensnowic, W., with Kudrjawzew, 666 (Hel.)
Schachow, S. D., with Orlowa, 514 (Z.)

Schadow, H., 1000 (Mal.)
Schaffle, K., with Colby, 232 (Hel.)
Schander, R. & Götze, G., 835 *bis* (Misc.)
Schapiro, L., 229 (Hel.)
—— & Cort, W. W., 174 (Hel.)
——, with Kritschewski & Grünbaum, 854 (Misc.)
Scharff, J. W., (611) (Mal.)
Schaumann, O., (280) (Dys.)
Schern, K., (925) (S.S.)
Schewelewa, E. M., with Wolsky, 132 (Mal.)
Schichobalowa, N. P., with Skrjabin & Podjapolskaya, 208 (Hel.)
Schilling, C., 59 (Misc.), 586 (Mal.), 894 (S.S.)
—— & Schreck, H., 484 *bis* (Z.)
Schipitsina, N. K., 512 (Z.)
Schjenti, A., 176 (Hel.)
Schlingman, A. S., 750 (Rab.)
Schlossmann, K., 333 (Lep.)
Schmelewa, A. A., 226 (Hel.)
Schmidt, B., with Sabolotnow, 394 (Pl.)
Schmidt, G. W., with Doerr, 208 (Hel.)
Schneider, H., 606 (Mal.)
Schneider, O., 650 (Lep.), (867) (Misc.)
Schöbl, O., 443, 444 *quat.*, 445 *bis* (Y. & S.)
—— & Garcia, O., 443 (Y. & S.)
——, Tanabe, B. & Miyao, I., 443 (Y. & S.)
Schockaert, J., 317 (R.B.F.)
Schoen, R., with Levaditi, Anderson & Selbie, 302 (R.F.)
——, with ——, Ravaut & Lépine, 937, 938 (C.Bu.)
Schoening, H. W., 748 (Rab.)
Schorlemmer, R., (1032) (Mal.)
Schotte, A., 839 (Misc.)
Schourenkova, A. & Nossina, V., 497 (Z.)
Schousboé, 968 (Oph.)
Schreck, H., with Schilling, 484 *bis* (Z.)
Schüffner, W., 164 (K.A.)
——, Dinger, J. E. & Snijders, E. P., 718 (Y.F.)
Schüffner, W. A. P., with —— & ——, 291, 295 (Y.F.)
——, with ——, —— & Swellengrebel, 290, 725 (Y.F.)
Schuhardt, V. T., with Chandler & Milliken, 217 (Hel.)
Schulman, E. S., with Skrjabin & Althausen, 683 (Hel.)
Schumaker, E., 496 *bis* (Z.)
Schürmann, K., with Mazza & Romaña, 923 (S.S.)
Schütz, F., 884 (Chl.)
Schwartz, A., Azam, A. & Yovanovitch, M., 181 (Hel.)
Schwartz, L. S., with Fradkin, 41 (Misc.)
Schweinburg, F., with Löffler, 745 (Rab.)
—— & Windholz, F., 246 (Rab.)
Schwetz, J., 493, 501, 503 (Z.), 585 (Mal.), 764 (Misc.)
—— & Baumann, H., 173 *bis*, 194 (Hel.)
——, —— & Cabu, F., 128 (Mal.)
Scott, E. L. & McKinley, E. B., 425 (Myc.)
Scott, G. W., (13) (Bl.), 121 (Mal.)
Scott, H., 622 (Fev.)
Scott, H. H., 28 (Misc.), 821 (Hist.)
Scott, J. A., 241 (Hel.)
Scriabine, K. I., Podyapolskaya, B. P. & Statirova, N. A., 182 (Hel.)
Scriver, J. B. & Waugh, T. R., 845 (Misc.)
Sebenzow, B. M., with Adowa, 511 (Z.)
Sebrell, W. H., with Goldberger, 473 (Pel.)
——, with ——, Wheeler & Rogers, 473 (Pel.)
Sédan, J., 477, 968 (Oph.)
Ségal, J., 604 (Mal.)
Selbie, F. R., with Levaditi, Anderson & Schoen, 302 (R.F.)
Self, P. A. W. & Corfield, C. E., (867) (Misc.)
Sellei, J., 448 (Y. & S.)
Semenza, C., 644, 645 (K.A.)
Semzova, O. M., 316 (Lept.), 735 (R.F.)
——, with Kritschewski & Ratner, 855 (Misc.)
Sen, A. K., 164 (K.A.)
Sen, A. N., 882 (Chl.)
Sen, P. B., with Brahmachari & Banerjea, 642 (K.A.)
Sen, R., 798 (Dys.)
Sena, J. A., with Adrougué, 327 (Lep.)
Senevet, G. & Champagne, R., 182 (Hel.)
——, with Sergent, Edm., Sergent, Et., Parrot, Foley & Catanei, 984 (Mal.)
Senior-White, R. A., 105 (Mal.)
Sequeira, J. H., 815 (Hist.)
Serebrennaja, A. I., with Palawandow, 245 *bis*, 741 (Rab.)
Sergent, A., with Sergent, Edm., Sergent, Et., Catanei & Trensz, 1013 (Mal.)
Sergent, Edm., Sergent, Et. & Catanei, A., 592 (Mal.)
——, ——, ——, Trensz, F. & Sergent, A., 1013 (Mal.)
——, ——, Parrot, L., Foley, H., Catanei, A. & Senevet, G., 984 (Mal.)
Sergent, Et. & Catanei, A., 599 (Mal.)
——, with Sergent Edm. & Catanei, 592 (Mal.)
——, with ——, ——, Trensz & Sergent, A., 1013 (Mal.)
——, with ——, Parrot, Foley, Catanei & Senevet, 984 (Mal.)
Serio, F., (801) (Am.)
Serpa, R., with Peña Chavarria & Bevier, 281 (Y.F.)
Serra, A., 665 (Hel.)
Serra, G., with van Nitsen, Lejeune, Miguens & Van den Branden, 439 (Y. & S.)
Seshadrinathan, N., 648 (K.A.)
——, with Vasudevan, 403 (Der.)
Sézary, A. & Roudinesco, 657 (Lep.)
——, with Vaudremer & Brun, 657 (Lep.)
Shah, K. S., with Yacob, 81 (Misc.)
Shaha, B., 880 (Chl.)
Shaha, B. B., 463 (Bb.)
Shanks, G., 462 (Bb.)
Shannon, R. C., 513 (Z.)
——, with Davis, 727 (Y.F.)
—— & Junior, J. S., (553) (Z.)
Shapiro, with Arnold, 435 (Chl.)
Shapland, C. D., with Doggart, 969 (Oph.)
Sharma, A. N., 171 (Hel.)
Sharp, E. A., 179 (Hel.)
Sharples, L. R., 54 (Misc.)
Shattuck, G. C. & Goodner, K., 447 (Y. & S.)
——, with Goodner, 773 (Misc.)
Shawki, I., 276 (Dys.)
Shelley, H. M., 465 (Pel.), 943 (Bl.)
Shepard, H. H., with Richardson, 509 (Z.)
Shetti, E. R., 479 (Oph.)

Shiha, M. M., 595 (Mal.)
Shillong, 560 (Lab.)
Shimokawa, H., with Chin-Kyu-Sui & Kamizawa, (866), (867) (Misc.)
Shircore, J. O., 12 (Bl.)
Shortt, 639 (K.A.)
Shortt, H. E., 578 (Mal.)
——, Smith, R. O. A. & Swaminath, C. S., 516 (Z.)
Shousha, A. T., 883 (Chl.)
Shrewsbury, J. F. D., 529 (Z.)
Shrivastava, D. L., with Hughes, 598 (Mal.)
Shute, P. G., with James & Nicol, 973 (Mal.)
Sicé, A., 351, 356, 903 (S.S.)
—— & Boisseau, R., 243 (Rab.), 379 (S.S.)
——, ——, Provost, J. & Deniel, 901 (S.S.)
Sidari, C., 1006 (Mal.)
Sieburgh, G., 1020 (Mal.)
da Silva, E. P., 747, 752 (Rab.)
Silva, F., 163 (K.A.), 401 (Der.)
—— & de Araujo, E., 165 (K.A.), 405 (Der.)
da Silva-Mello, A., (280) (Dys.)
Silvan, P., 44 (Misc.)
da Silveira, F., with Belfort, 856 (Misc.)
Silverman, D. N. & Feemster, R., 797 (Dys.)
Simic, T., 270, 793 (Am.)
Simmons, J. S., St. John, J. H. & Reynolds, F. H. K., 114 (Mal.), 613 (B.R.)
——, with —— & ——, 507 (Z.)
Sinclair, B. A., 635 (Fev.)
Singh, J., 141, 1025 (Mal.)
——, with Sinton & Harbhagwan, 587 (Mal.)
Sinha, H. K., (438) (Chl.)
Sinjuschina, M. N., with Kritschewski, 737 (R.F.)
Sinton, J. A., 517, (553) *bis* (Z.), 577, 596, 598, (1032) (Mal.)
——, Harbhagwan & Singh, J., 587 (Mal.)
—— & Kehar, N. D., 508 (Z.), 1024 (Mal.)
—— & Mulligan, H. W., 146 (Mal.)
Sioe, K. T., 169 (Hel.)
Sircar, B. L., with Raju, (438) (Chl.)
Skliar, N. & Rjabowa, M., 594 (Mal.)
Skrjabin, K. I., Althausen, A. J. & Schulman, E. S., 683 (Hel.)
——, Podjapolskaya, W. P. & Schichobalowa, N. P., 208 (Hel.)
Skrjabin, K. J. & ——, 675 (Hel.)
Slot, J. A., (740) (Lept.), 849 (Misc.)
Smirnow, G. G., 175 (Hel.)
Smith, E. C., 48, 838 *bis* (Misc.), 294 (Y.F.), 398, 411 *bis*, 412 (Der.), 441 (Y. & S.)
Smith, G. D., with Baumgartner, 661 (Sp.)
Smith, H. F., 713 (Y.F.)
Smith, H. M., 955 (Lep.)
Smith, O. A., with Phelps, Carroll, Washburn & Beagley, 215 (Hel.)
Smith, R. O. A., with Shortt & Swaminath, 516 (Z.)
Smith, S., 750 (Rab.)
Smyly, H. J., 277 (Dys.)
Smyth, F. G. A. & Cameron, W. M., 102 (Fev.)
Snijders, E. P., 619 (Fev.), 1000 (Mal.)
—— & Dinger, J. E., 728 (Y.F.)
——, with —— & Schüffner, 291, 295 (Y.F.)
——, with ——, ——— & Swellengrebel, 290, 725 (Y.F.)
——, with Schüffner & Dinger, 718 (Y.F.)
Snyder, J. W., 788 (Am.)
Sobrinho, J. M., with Rezende, 936 (C.Bu.)
Soeriobroto, M. A., 695 (B.R.)
Soesilo, R., with de Rook, (552) (Z.)
Soewandi, (280) (Dys.)
Sofieff, M. S., 300 (R.F.)
———, with Chodukin, 155 (K.A.)
Soga, A. R. B., 768 (Misc.)
Sohier, R., with Jausion, 416 (Myc.)
Sollier, N., with Dubois, 87, 704 (Und.)
Solowjewa, J. W., with Glusman, 746 (Rab.)
——, with —— & Predtetschenskaja, 244 (Rab.)
——, with —— & Sadowsky, 244 (Rab.)
Sommermeyer, V., with Holder & Osborne, 791 (Am.)
Sondag, A. V. A., 773 (Misc.)
Sophiew, M. S., with Chodoukine & Chewtchenko, 641 (K.A.)
Sorapure, V. E., with Armand-Delille & Hurst, 56 (Misc.)
Sorge, G., 854 (Misc.)
Soromenho, L., 947 (Bl.)
Sorour, M. F., 190 *bis*, 193 (Hel.)
Souchard, with Letonturier & Martin, 40 (Misc.)
South African Institute for Medical Research, 557 (Lab.)
Southern Medical Journal, 108, 976 (Mal.)
de Souza-Araujo, H. C., 656 *bis*, 657 (Lep.)
Spain, 83 (B.R.)
Speierer, C., 139, 146 (Mal.)
Spencer, H. H., (1032) (Mal.)
Spero. L. P., 886 (H.S.)
Spindler, L. A., with Otto, 174 (Hel.)
Spoto, F. & Ruffino, P., 957 (Lep.)
Spyropoulos, N. J., 617 (Fev.)
Ssyssine, A., (395) (Pl.)
Stalder, H. & Lauterburg, M., (867) (Misc.)
Stannus, H. S., 31 (Misc.)
Starr, P. & Gardner, L., 663 (Sp.)
Statirova, N. A., with Scriabine & Podyapolskaya, 182 (Hel.)
Staudt, 192 (Hel.)
Steel, C. R., 584 (Mal.)
van Steenis, P. B., 634 (Fev.), 798 (Dys.)
Steevenson, G. F., 254 (Rab.)
Stefanopoulo, G. J. & Codounis, A., 295 (Y.F.)
——, with Fourneau, Tréfouel, M. & Mme., Benoit, de Lestrange & Melville, 64 (Misc.)
Stein, A. A., 959 (Lep.)
Stein, A. K., with Pawlowsky, 534 (Z.)
Steinberg, B., 41 (Misc.)
Stekhoven, J. H. S., Jr., 666 (Hel.)
Stender, A., with Müller, 191 (Hel.)
Stephens, E. D., with Dunbar, 276 (Dys.)
Stephens, J. W. W., 13 (Bl.)
Sterkin, E. J., 142 (Mal.)
Stettner, E., 275 (Dys.)
Steven, G. H., 565 (Lab.)
Stewart, A. D., 779, 869 (B.R.), 985 (Mal.)
Sticker, G., 827 (Hist.)
Stiles, C. W., 228 (Hel.)
Stitt, E. R., 803 (Hist.)
Stiven, H. E. S., 36 (Misc.)
Stoel, G., 244 (Rab.)
Stolnikoff, W. I., 254 (Rab.)
Storrs, R., 808 (Hist.)
Storti, E., 201 (Hel.)

Stoute, D. G., with Wildish, 653 (Lep.)
Stradomsky, B. N., Petrowsky, I. N., Popow, W. W. & Rudnew, G. P., 142 (Mal.)
Straub, M., 18, 772 (Misc.)
Strauss, A. E., with Eyermann, 133 (Mal.)
Strickland, C., 82 (Misc.)
—— & Chowdhury, K. L., 943, 945 (Bl.)
—— & Roy, D. N., 1018 (Mal.)
Strom, J., (242) (Hel.)
Struthers, E. B., 637 (K.A.)
Stuart, G. & Krikorian, K. S., 253 (Rab.)
Stubbs, W. D., with Wilkinson, Desai & Beharilal, 988 (Mal.)
Stumberg, J. E., 240 (Hel.)
Stylianopoulo, M., with Mélanidi, 617 (Fev.)
Suarez, J., 215 (Hel.)
Suarez, R. M., 195 (Hel.), (664) (Sp.)
Suarez de Puga, L. & Colomo de la Villa, G., 631 (Fev.)
Sun, T. C. Y. & Young, M. P., 988 (Mal.)
Surbek, K. E., 19 (Misc.), (280) (Dys.), 998, 1014 (Mal.)
Susman, W., 470 (Pel.)
Swaminath, C. S., with Shortt & Smith, 516 (Z.)
Swanidse, D., 176 (Hel.)
Swarup, A., 468 (Pel.)
Sweet, W. C., 170, 231, (242) (Hel.)
Swellengrebel, N. H., 981 (Mal.)
—— & de Buck, A., 1018 (Mal.)
——, with Dinger, Schüffner & Snijders, 725 (Y.F.)
——, with ——, —— & Snijders, 290 (Y.F.)
Sylvanus, D., with Aalsmeer, 460 (Bb.)
Symes, C. B., 984 (Mal.)
Szidat, L. & Wigand, R., 671 (Hel.)

T

Taddia, L., with Franchini, 732 (R.F.)
Takahashi, S., 184 (Hel.)
——, Ishikawa, K., Ogawa, S. & Ida, T., 467 (Pel.)
Takeuchi, Y., 887 (H.S.)
Takino, M., 327 (Lep.)
Talbot, 965 (Oph.)
Talec, 951 (Bl.)
Taliaferro, W. H., 977 (Mal.)
——, with Cannon, 494 (Z.)
—— & Hoffman, W. A., 214 (Hel.)
Talice, R. V., (307) (R.F.), 416, 420 (Myc.), 535 (Z.)
Talysin, T., 201 (Hel.)
Tamalet, E., 596 (Mal.)
Tamiya, T., with Nagayo, Mitamura & Sato, 635 (Fev.)
Tanabe, B., with Schöbl & Miyao, 443 (Y. & S.)
Tanaka, K., Kaiwa, J., Teramura, S. & Kagaya, J., 103 (Fev.)
Tanikawa, K., with Morishita, Namikawa & Matsuura, (1032) (Mal.)
Tanon, L., Cambessédès, H. & Decourt, P., (1032) (Mal.)
Tanzer, F., 59 (Misc.)
Tao, S. M., 487 (Z.)
Taramelli, 406 (Der.)
Tarassoff, S., (740) (Lept.)
Tarassow, W., 667 (Hel.)
Tarayre, 453 (C.Bu.)
Tate, P., (415) (Der.)
Taute, M., with Manteufel, 427 (B.R.)
Taylor, A. W., 367 (S.S.), 501, 519 (Z.)
—— & Lester, H. M. O., 368 (S.S.)
Taylor, H. W. Y., 639 (K.A.)
Taylor, J., with Asheshov, Morison, Malone & Iyengar, 863 (Misc.)
—— & Goyle, A. N., 739 (Lept.)
——, Greval, S. D. S. & Thant, U., 273 (Dys.)
—— & Menon, K. N., 248 (Rab.)
Taylor, K. P. A., 27 (Misc.), 107, 109 (Mal.), 397 (Der.)
Tchakirian, A., with Levaditi, Bardet & Vaisman, 908 (S.S.)
Tedeschi, C., (664) (Sp.)
Tegoni, G., with Aureli, B. W., 120 (Mal.)
Teitel, D., (280) (Dys.)
Teixeira, J. de C., with Villela, 240 (Hel.)
Teramura, S., with Tanaka, Kaiwa & Kagaya, 103 (Fev.)
Tessitore, with Marone, 845 (Misc.)
Tetsumoto, S., 437 (Chl.)
Thant, U., with Taylor & Greval, 273 (Dys.)
Thapar, G. S., 170 (Hel.)
Theiler, M., 723 (Y.F.)
——, with Yang, 314 (Lept.)
Theobald, G. W., 31 (Misc.), 117 (Mal.)
Theodor, O., with Adler, 151, 152, 153, 639, 640 (K.A.)
——, with —— & Lourie, 516 (Z.)
Theodore, J. H., 161 (K.A.)
van Thiel, P. H., 512, 535, 536 (Z.)
Thim, J. R., 857 (Misc.)
Thiroux, (395) (Pl.)
Thiry, U., with Bessemans, 311 (Lept.)
Thomas, A. D. & Jackson, C., 744 (Rab.)
Thomas, L. J., 199 (Hel.)
Thompson, C. J. S., 815 (Hist.)
Thompson, T. O. & Grant, P. F. A., 121 (Mal.)
Thomson, D. F., 547 (Z.)
——, with Kellaway, 547 (Z.)
Thomson, J. G., 123 (Mal.)
Thomson, J. W., 48 (Misc.)
Thonnard-Neumann, E., 72 *bis* (Misc.)
——, Camacho-Moya, J. & Brewster, K. C., 402 (Der.)
Thornton, E. N., 380 (Pl.), 766 (Misc.)
Thurber, D. S., 703 (Und.)
Tikhomirova, M. & Nikanorov, S., 393 (Pl.)
Tilden, E. B. & Tyler, J. R., 475 (Oph.)
Timofeewa, M. E., 151 (K.A.)
Timpano, P., 149, 157 (K.A.), 738 (R.F.)
Tinker, I. S. & Rudnew, G. P., 390 (Pl.)
Tisseuil, J., 326 *ter*, (658), 956, 957, (964 *bis*) (Lep.)
Tjokronegoro, R. M. H., 858 (Misc.)
Todd, K. W., 929 (Y. & S.)
Tohyama, Y. & Yasukawa, Y., 437 (Chl.)
Tolosa, A., 958 (Lep.)
Tomb, J. W., 433 (Chl.)
—— & Maitra, G. C., 885 (Chl.)
Tomita, S., (740) *ter* (Lept.)
Tonnet, J., with Loeper, 679 (Hel.)
Toporkow, F. M., 994 (Mal.)
Torgerson, W. R., 73 (Misc.)
Torpy, C. D., 744 (Rab.)

Torres, C. M., 294 *bis* (Y.F.)
—— & Villela, E. L., 223 (Hel.)
Toscano, C., 602 (Mal.)
Toullec, F., 37 (Misc.), 96 (Fev.)
——, with Blanchard, 898 (S.S.)
—— & Riou, (964) (Lep.)
Toumanoff, C., with Roubaud, 507, 509 (Z.)
Tournier, (801) (Am.)
Tôyama, I., Miyoshi, S. & Kubokawa, K., 332 (Lep.)
Toyoda, H., 738 (R.F.)
Trabaud, J., 131, 993 (Mal.), 159 (K.A.) 621, 622 (Fev.)
Tran-Van-Manh, with Chesneau, (241) (Hel.)
Traum, E. & Linden, H., 917 (S.S.)
Tréfouel, M. & Mme., with Fourneau & Benoit, 64 (Misc.)
——, with ——, Stefanopoulo, Benoit, de Lestrange & Melville, 64 (Misc.)
Trensz, F., with Sergent, Edm., Sergent, Et., Catanei & Sergent, A., 1013 (Mal.)
Trincao, C., 331 (Lep.)
Troisier, J. & Boquien, Y., 309, 311 (Lept.)
—— & Deschiens, R., 209 (Hel.)
Tscherikower, R. S., 855 (Misc.)
Tsiminakis, K., 93 (Fev.)
Tskimanauri, G., 601 (Mal.)
Tsuchiya, H., 488 *bis* (Z.)
—— & Andrews, J., 488 (Z.)
—— & Mizushima, H., 488 (Z.)
Tsykalas & Riegl, R., 190 (Hel.)
Tubangui, M. A., 675 (Hel.)
—— & Francisco, S. A., 199 (Hel.)
Tuchtan, D., with Dalma, 605 (Mal.)
Turner, A. W., 858 (Misc.)
Turner, W. H., Jr., 637 (K.A.)
Twining, C. N., 688 (Hel.)
Twitchell, E. W., 138 (Mal.)
Tyler, J. R., with Olitsky, 965 (Oph.)
——, with—— & Knutti, 966 (Oph.)
——, with Tilden, 475 (Oph.)
Tyner, J. D., 660 (Sp.)

U

Uhlenhuth, P., 308 (Lept.)
Ujlaki, P., 950 (Bl.)
Ulmi, P., 11 (Bl.)
Umansky, J., 1015 (Mal.)
Union of South Africa, 582 (Mal.), 649 (Lep.)
United Fruit Company, Boston, 106, 978 (Mal.)
Urbain, A., 435 (Chl.)
——, with Marie, 256, 746 (Rab.)
Urueña, J. G., 398 (Der.)
Usher, B., 957 (Lep.)
——, with Burgess, 413 (Der.)
Utkina-Ljubowzewa, X., with Zeiss, 363 (S.S.)

V

Vaccarezza, R. F., (801) (Am.)
Vaidya, S. K., (664) (Sp.)
Vaisman, A., with Levaditi, Bardet & Tchakirian, 908 (S.S.)
Valerio, A., (801) (Am.)
Valsamaki, A., with Pédaros & Caragianopoulos, 628 (Fev.)
Valtis, J., with Blanc, (648) (K.A.)
Van den Branden, F., 138 (Mal.), 355 (S.S.), (964) (Lep.)
——, Dumont, P. & Nélis, P., 305 (R.F.), 900 (S.S.)
——, with van Nitsen, Lejeune, Miguens & Serra, 439 (Y. & S.)
Van der Hoeven, J., 805 (Hist.)
Van der Made, W. M., 337 (B.R.)
Vanni, V., 852 (Misc.)
Vargas, A., 119 (Mal.)
Vassiliadis, P., 371 (S.S.), 543 (Z.)
—— & Jadin, J., 306 (R.F.), 370 (S.S.)
Vasudevan, A., with Cherian, 422 (Myc.)
—— & Seshadrinathan, N., 403 (Der.)
Vaucel, 127 (Mal.)
Vaucel, M., 368 (S.S.)
—— & Boisseau, R., 796 (Dys.), 905 (S.S.)
Vaudremer, A., Sézary, A. & Brun, C., 657 (Lep.)
de Vault, V. T., with Makel, 221 (Hel.)
Vedder, E. B., 324 (Lep.)
—— & Feliciana, R. T., (464) (Bb.)
—— & Masen, J. M., 1002 (Mal.)
Védrenne, 626 (Fev.)
Vejdovsky, V., 967 (Oph.)
Vellard, J., with Brasil, 540 (Z.)
—— & Penteado, J., 551 (Z.)
—— & Vianna, M., 551 *bis* (Z.)
Velu, H., Balozet, L. & Zottner, G., 734, 736 *ter*, 738 (R.F.)
Venezuela, 697 (B.R.)
Venturi, L. C., 264 (Am.)
de Vera, B., 322 (Lep.)
Verge, J. & van Huyen, P., 255 (Rab.)
Vergeer, T., 678 (Hel.)
Verschaffelt, F., with Dinger, 314 (Lept.)
Veterinary Bulletin, 614 (B.R.)
Viala, J., 250, 749 (Rab.)
——, with Nicolau & Kopciowska, 246 (Rab.)
Vialatte, C. & Remontet, E., 140 (Mal.)
—— & Sainte-Marie, P. E. F., 994 (Mal.)
Vianna, M. & Pedro, A., 457 (Bb.)
——, with Vellard, 551 *bis* (Z.)
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